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Radiology of the Pharynx and the Esophagus

With Contributions by

K. Aksglæde · A. Ba-Ssalamah · J. E. Benson · M. Bülow · M. Y. Chen · C. Czerny
D.-M. Denk · E. Eisenhuber · O. Ekberg · P. Funch-Jensen · C. Hannig · M. S. Levine
B. Martin-Harris · G. Nylander · R. Olsson · D. J. Ott · B. C. Pineau · P. Pokieser
S. Rubesin · M. Scharitzer · W. Schima · R. Schöfl · P. Thommesen · A. Vossough
A. Wuttge-Hannig

Foreword by

A. L. Baert

With 275 Figures in 477 Separate Illustrations, 28 in Color and 17 Tables

Springer

Olle Ekberg, MD, PhD
Professor, Department of Diagnostic Radiology
Malmö University Hospital
20502 Malmö
Sweden

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Foreword

It is well known that in the Western part of the world and in Japan, the population is aging rapidly, mainly due to advances in healthcare. Swallowing disturbances and dysphagia are therefore becoming increasingly common problems in the daily practice of many radiologists.

This volume very comprehensively presents all imaging aspects of the pharynx and the esophagus. It has been written and edited from a clinical point of view, beginning with anatomy and physiology and covering the whole spectrum of functional and morphological disorders of these organs.

Great attention is given to the various examination techniques now used, with reference made to manometric and endoscopic features where appropriate.

The text is well written and is supplemented by numerous superb illustrations.

The editor, Professor Dr. O. Ekberg, is an internationally acclaimed expert in the field with a lifelong dedication to and interest in imaging of the pharynx and esophagus. The authors of the individual chapters have been invited to contribute to the volume on account of their long-standing experience and major contributions to the radiological literature on the topic. I would like to thank the editor and the authors and congratulate them most sincerely on this outstanding volume.

I am confident that it will meet with great interest both from general and gastrointestinal radiologists and from surgeons, ear, nose, and throat specialists, and gastroenterologists, and I am sure that it will be as well received by readers as previous volumes published in this series have been.

Leuven

Albert L. Baert

To my wife

Guje

for her love, encouragement and support during all those years

Olle Ekberg

Preface

Accurate radiologic assessment of the pharynx and esophagus is crucial for diagnosis and treatment of patients with dysphagia. Such a work-up often relies on a multi-disciplinary approach. Radiology is always crucial, since transportation from ingestion until the bolus reaches the stomach is best monitored with radiology. The diagnosis relies on an interdisciplinary approach; for the radiologist this means that he must have a thorough clinical understanding of the patient's symptomatology and disease entities. It also means that the clinician, whether an ENT-surgeon, neurologist, gastroenterologist or speech-and-language pathologist, must have a profound understanding of the potentials and limitations of radiologic studies. This book is an attempt to cover some of this territory, by presenting important aspects of radiology in patients with dysphagia.

A thorough knowledge and understanding of anatomy, physiology, pathophysiology, pathology and clinical background is required for those using radiology as a modality for dysphagia evaluation and treatment. I have selected topics that represent problems most commonly encountered in these clinical settings to provide an educational and reference tool for a broad readership. The text provides both theoretical background and practical hints for understanding how radiology can contribute in the dysphagic patient. The volume spans the clinical scenario from subtle, mild dysphagia supposedly due to an isolated neuritis, on the one extreme, to patients who have undergone esophagectomy or other major surgical procedures, on the other.

This work is truly a compilation of the experience culled from different experts. Therefore both techniques for performing studies and interpretation may differ simply as a reflection of personal experience. As long as radiology of the pharynx and esophagus is not done cursorily, different techniques can be used. However, custom tailoring means different things to different people. Remember that the three different anatomical and functional compartments (oral cavity, pharynx and esophagus) are very different in terms of neurologic control and panorama of disease entities. It is hoped that this volume may provide thorough and ample information to all those engaged in the imaging evaluation of swallowing.

Both my own interest and this book grew out of contacts and interaction with several people over the years. I am particularly grateful to have been able to spend days and weeks together with Dr James F. Bosma. His encyclopaedic knowledge about the normal and abnormal swallow and his ability always to put radiology in a larger context was pivotal in my education.

I also owe a debt of gratitude to Dr Göran Nylander, who opened my eyes to this fascinating topic. My inspiration came from his teaching.

Moreover, I learned tremendously from endless discussions with Dr Michael J Feinberg, who had the ability to see beyond the milliseconds and to visualise how to conceptually understand swallowing. He thought and taught in terms of morphodynamics.

With deep appreciation I must confess that Eva Prahl, with her skilful assistance, kept all the loose ends together for this volume.

Malmö

Olle Ekberg

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1 Anatomy and Physiology

Olle Ekberg and Göran Nylander

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1.1 Introduction

The swallowing apparatus is made up of three anatomically and functionally separated, but integrated, areas, namely the oral cavity, the pharynx and the esophagus. These are tubular structures with muscular walls, in certain areas containing bone and cartilage. Each compartment functions independently, but for a successful swallowing process a finely tuned coordination between the compartments is necessary. Each compartment acts as a hydrodynamic pump. Between these pumps are interconnected valves.

In order to interpret the radiological examination, detailed knowledge about anatomy and physiology in this area is mandatory. In this context it is also important to understand that the larynx, both anatomically and physiologically, is an integrated part of the pharynx during swallowing. The nomenclature used in this chapter corresponds to anglicised Latin commonly in use (Williams et al. 1989). The description below refers to the adult individual. Those interested in newborns and infants are referred to works by Bosma (1973, 1976).

O. Ekberg, MD
Professor, Department of Diagnostic Radiology, Malmö University Hospital, 205 02 Malmö, Sweden
G. Nylander, MD
Associate Professor, Department of Diagnostic Radiology, Malmö University Hospital, 205 02 Malmö, Sweden

1.2 Anatomy of the Pharynx and Larynx

1.2.1 Cartilages of the Larynx and Pharynx

Several of the important swallowing muscles insert on the inside of the mandible (Fig. 1.1). On the inside and medial surface of the mandible there is a centimetre-sized crest called the mylohyoid line, where the mylohyoid muscle inserts. Anteriorly in the midline on the posterior surface of the mandible there is a couple of eminences (mental spines) on which the geniohyoid and genioglossus muscles insert. These muscles then also insert within the tongue and on the hyoid bone, respectively. The hyoid bone is made up of a body and four horns, two on each side (Fig. 1.2). The upper two are called: the lesser cornu of the hyoid bone, and the lower ones: the greater cornu of the hyoid bone. From the upper lesser cornu there is a ligament that connects the cornu with the styloid process of the skull base. This ligament is called the stylohyoid ligament.

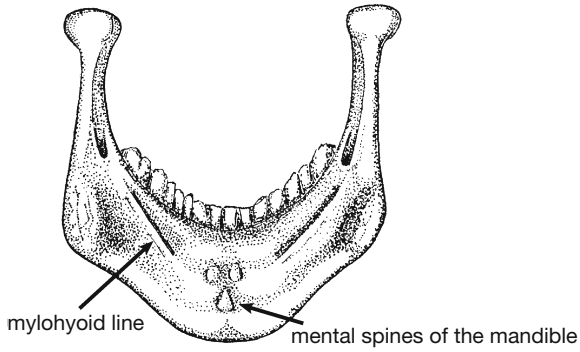

Fig. 1.1. The mandible seen from posteriorly

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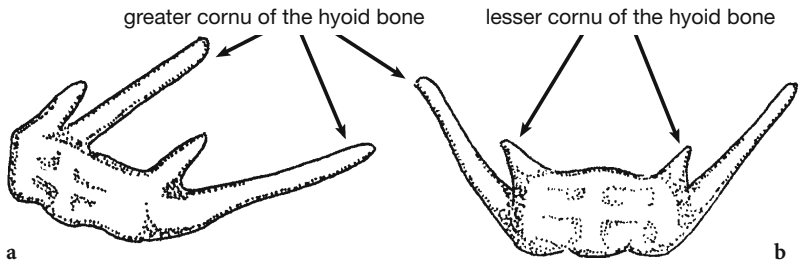

Fig. 1.2a,b. The hyoid bone seen from superiorly and the left (**a**) and from anteriorly (**b**)

The thyroid cartilage is made up of two quadrilateral laminae, their anterior borders fused inferiorly and with a convexity superiorly and anteriorly. It also has a notch in the midline and superiorly. Posteriorly the cartilage has four horns (cornu). Two of these have a superior direction (superior cornu of the thyroid cartilage) and two an inferior direction (inferior cornu of the thyroid cartilage) (Fig. 1.3).

The superior cornu is via the thyrohyoid ligament connected with the greater cornu of the hyoid bone. The inferior cornu articulates directly against the cricoid cartilage. The hyoid bone and the thyroid cartilage are connected, not only with the medial and lateral thyrohyoid ligaments but also by the thyrohyoid membrane (Fig. 1.4). There is a lateral opening in the thyrohyoid membrane through which the laryngeal artery, vein and nerve pass. There is also a small cartilage in the posterior and lateral part of the thyrohyoid ligament. This is called the triticeal cartilage.

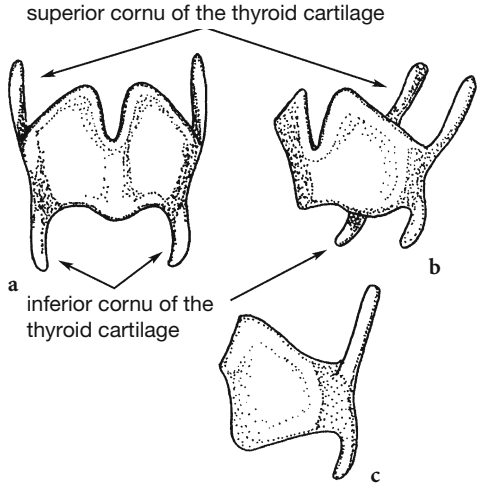

Fig. 1.3a–c. The thyroid cartilage seen from anteriorly (**a**), from anteriorly and left (**b**) and from the left (**c**)

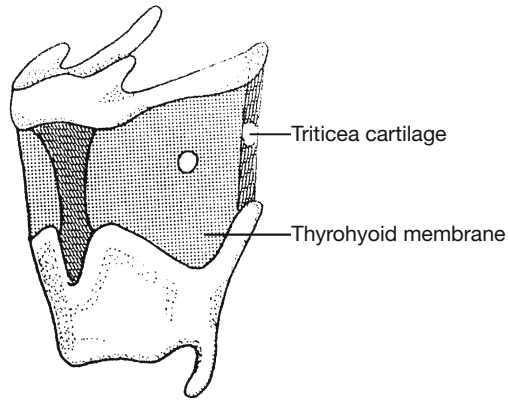

Fig. 1.4. Hyoid bone and thyroid cartilage seen from anteriorly and the left. *Light hatching*, the thyrohyoid membrane. *Dark hatching*, the thyrohyoid ligament lateral and medial. There is a hole in the membrane for the passage of vessels and nerves

The cricoid cartilage has the shape of a signet ring and is made up of a thin anterior part called the arcus of the cricoid cartilage and a posterior thicker portion called the lamina of the cricoid cartilage (Fig. 1.5). On its lateral margin the cricoid cartilage has an articulate facet for the inferior cornu of the thyroid cartilages. The lamina continues superiorly and dorsally in an eminence that ends with an articulate facet. Against this surface the arytenoid cartilages articulate. Two inferior horns of the thyroid cartilage articulate as described above against the cricoid cartilage. The thyroid and cricoid cartilages are also connected via the cricothyroid ligament (Fig. 1.6). Inferiorly to the cricoid cartilage is the trachea.

The core of the epiglottis is made up of cartilage. This thin foliate lamella has the form of a racket with a plate and a shaft. The shaft (petiolus), has a ligament (thyroepiglottic ligament) that connects it to the posterior surface of the thyroid cartilage (Fink and Demarest 1978) (Fig. 1.7a).

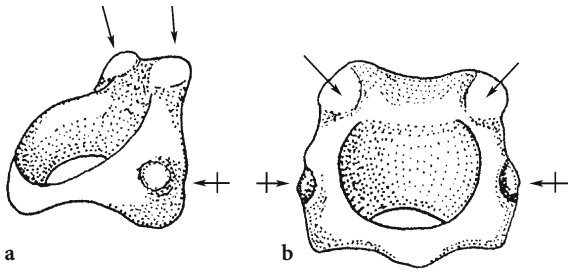

Fig. 1.5a,b. The cricoid cartilage seen from the left (**a**), and from anteriorly (**b**). There are two articulate surfaces for the arytenoid cartilages (*plain arrows*). There are also articulate surfaces for the cricoid cornu of the thyroid cartilage (*crossed arrows*)

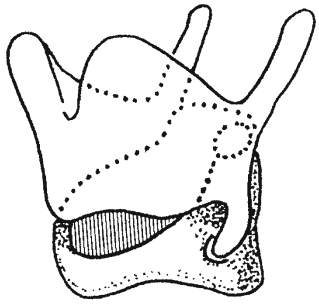

Fig. 1.6. The thyroid cartilage and cricoid cartilage with the cricothyroid ligament (*shaded*)

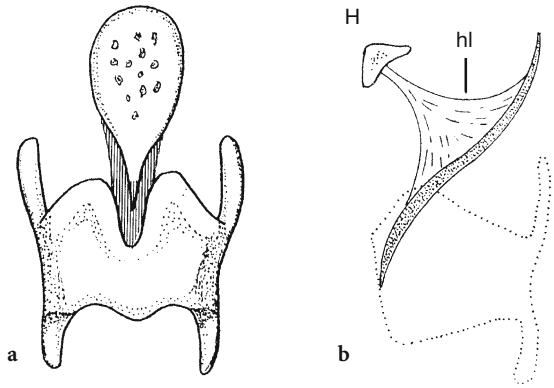

Fig. 1.7a,b. a The thyroid cartilage and epiglottis (seen from anteriorly) are connected with the thyroepiglottic ligament. **b** The hyoid bone (*H*) (seen from the *left*) is connected to the epiglottis with the hyoepiglottic ligament (*hl*)

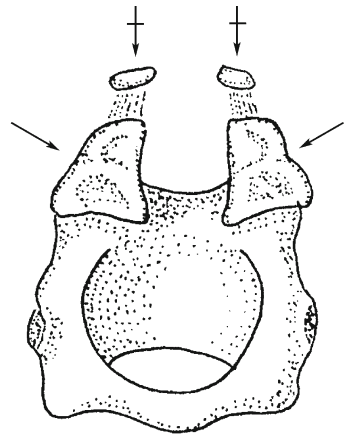

Fig. 1.8. Cricoid cartilage (seen from anteriorly). The arytenoid cartilages (*plain arrows*) and corniculate cartilages (*crossed arrows*) are located on top

The anterior surface of the epiglottis has a fan-shaped ligament connecting it to the hyoid bone (Fig. 1.7b). This ligament is an extension of the median glossoepiglottic ligament.

The arytenoid cartilages are shaped like small pyramids and are located at the posterior and superior corners of the cricoid cartilage. On top of this pyramid is another small cartilage, namely the corniculate cartilage (Fig. 1.8).

Thereby there is a wall of cartilages, ligaments and membranes extending from the hyoid bone and inferiorly. It reaches all the way to the anterior surface of the trachea. In the following the relation of musculature and mucous membrane to these stabilising structures will be described.

1.2.2 Muscles

The floor of the mouth is made up of several muscles, the position of which are given in Figures 1.9 and 1.10. The caudal extreme of the floor of the mouth is made up of the geniohyoid muscle and mylohyoid muscle. The latter inserts on the mylohyoid line on the mandible. It extends to the hyoid bone where it inserts (Fig. 1.9). It is made up of a broad muscular diaphragm that covers most of the floor of the mouth. Covering this muscle is the geniohyoid muscle extending from the mental spines in the midline of the mandible to the body of the hyoid bone. The stylohyoid muscles extend from the styloid process to the lesser cornu on both sides (Fig. 1.10). Inferiorly are the thyrohyoid muscle, the hyoid bone and the thyroid cartilage (Fig. 1.9). Inferior to the hyoid bone is the sternohyoid muscles and omohyoid muscles.

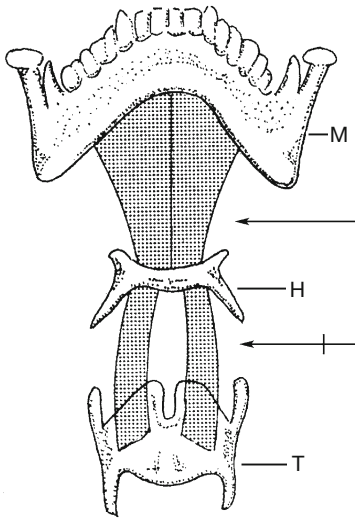

Fig. 1.9. The mandible (*M*), hyoid bone (*H*) and thyroid cartilage (*T*) seen from anteriorly. The mylohyoid (*plain arrow*) and thyrohyoid (*crossed arrow*) muscles are indicated

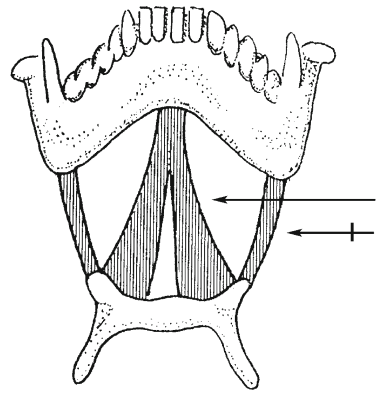

Fig. 1.10. The mandible and hyoid bone seen from below and anteriorly. The geniohyoid (*plain arrow*) and stylohyoid (*crossed arrow*) muscles are indicated

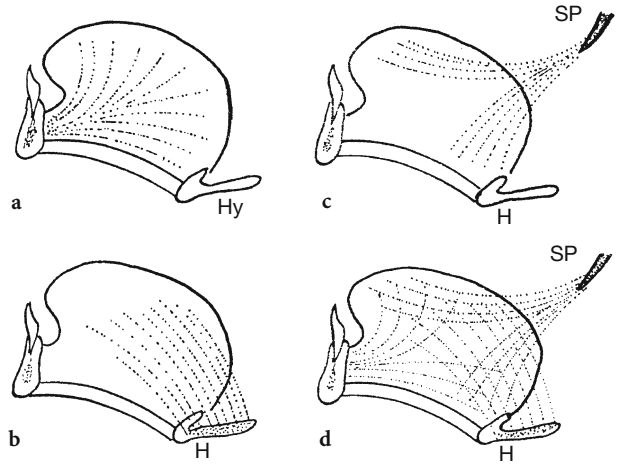

Fig. 1.11a–d. The tongue musculature, hyoid bone (*H*), and styloid process (*SP*). **a** Genioglossus muscle, **b** hyoglossus muscle, **c** styloglossus muscle, **d** composite drawing of the three muscles shown in **a–c**

1.2.2.1 Muscles of the Tongue

The genioglossus muscle is the largest muscle of the tongue and it extends from the mental spines on the mandible. This fan-shaped muscle widens as it extends backwards into the tongue. The superior fibres run to the tip of the tongue, and the middle fibres to the dorsum of the tongue and a few of the inferior fibres extend to the hyoid bone where the muscle inserts on the body of the hyoid bone (Fig. 1.11a). The hyoglossus muscle extends from the body and greater cornu of the hyoid bone and extends from there superiorly into the lateral portions of the tongue (Fig. 1.11b). The styloglossus muscle extends from the styloid processes of the skull base and the stylomandibular ligaments. It then extends into the lateral part of the tongue all the way to the tip of the tongue (Fig. 1.11c).

These three muscles join within the tongue and the muscle bundles fuse (Fig. 1.11d).

There are also a couple of external tongue muscles that connect the tongue with the skull base, the mandible and the hyoid bone. Other tongue muscles are separated from the above and located solely within the tongue. They can be divided into four: (1) the longitudinal, superficial muscle, (2) the longitudinal deep muscle, (3) the transverse lingual muscles and (4) the vertical lingual muscles. A small portion of the transverse lingual muscles runs up into the soft palate where it is called the glossopalatinus muscle (Fig. 1.12). Another small portion of this muscle is called the glossopharyngeal muscle and extends into the pharyngeal wall musculature (Fig. 1.12). In this way the musculature of the tongue inserts on the skull base, mandible, hyoid bone, soft palate and lateral pharyngeal wall.

1.2.2.2 Muscles of the Soft Palate

The soft palate has an important function during swallowing. It is made up of a fibrous aponeurosis on which a couple of swallowing muscles insert. The levator veli palatinum muscle extends from the inferior and lateral surface of the temporal bone close to

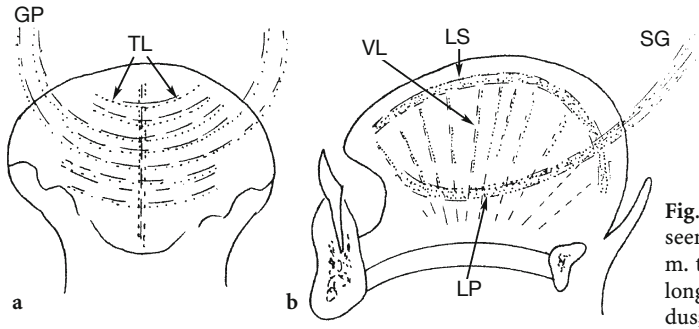

Fig. 1.12a,b. Internal tongue musculature. The tongue seen from anteriorly in (**a**) and from the left in (**b**). *TL*, m. transversus linguae; *VL*, m. verticalis linguae; *LS*, m. longitudinalis superficialis; *LP*, m. longitudinalis profundus; *GP*, m. glossopharyngeus; *SG*, m. styloglossus

the foramen of the internal carotid artery as well as from the inferior aspect of the tubal cartilage (of the tuba auditiva). The muscle then extends inferiorly, medially and anteriorly and inserts on the midportion of the aponeurosis of the soft palate (Fig. 1.13). The tensor veli palatini muscle extends from the skull base and from the pterygoid processes of the sphenoid bone and extends first inferiorly and then turns at a right angle medially over the hamulus of the processus pterygoideus to spread horizontally in the aponeurosis of the soft palate (Fig. 1.14).

The palatopharyngeal muscle is the most prominent muscle in the soft palate and constitutes the arch form. It extends from the inferior body of the tubal cartilage, pterygoid processes and aponeurosis of the soft palate. This is the posterior extreme of the soft palate. The muscle then extends further inferiorly and posteriorly and forms part of the posterior wall of the pharynx. It also reaches the posterior surface of the thyroid cartilage (Fig. 1.15).

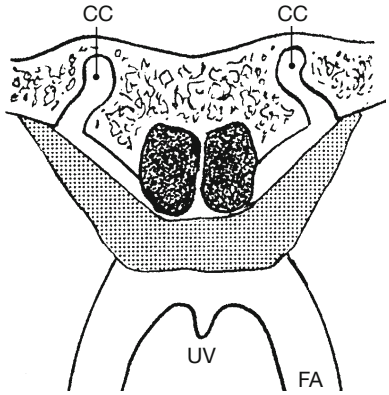

Fig. 1.13. Muscles of the levator veli palatini (*shaded*). The picture shows the skull base with choanae (*dark*) as well as the canalis caroticus (*CC*). The uvula (*UV*), as well as the faucial arcs (*FA*), are indicated

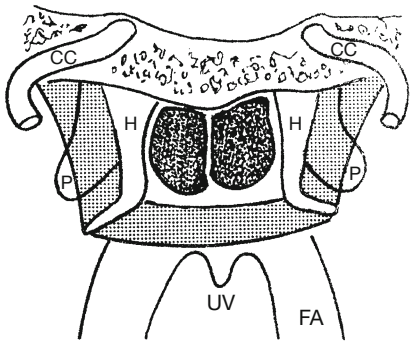

Fig. 1.14. Tensor veli palatini muscle (*shaded*). The picture shows the skull base with choanae (*dark*) as well as canalis caroticus (*CC*). Pterygoideus process (*P*) and hamulus of the processus pterygoideus (*H*) is indicated, as well as the uvula (*UV*) and the faucial arcs (*FA*)

1.2.2.3 Muscles of the Pharynx

All muscles in the oral cavity, larynx and pharynx are striated. Of the two arches that surround the tonsils the medial is made up of the previously described palatopharyngeal muscle and the lateral of the glossopalatinal muscle (Fig. 1.16).

The walls of the pharynx are made up of a fibrous fascia connected to the mucosa on the inside and to the musculature on the outside of the wall. Superiorly towards the skull base there is no proper muscular layer. The only layers here are the mucosa and fascia called the fibrous layer of the pharynx. This has a width of about 2 cm. Further inferiorly is the constrictor musculatures (Fig. 1.17). The main part of the wall of the pharynx is made up of constrictor muscles and elevators. The elevators are located on the inside which is unique in the gastrointestinal tract. The muscles surrounding the oropharyngeal junction area are schematically drawn in Figure 1.18.

The pharyngeal constrictors are made up of three portions. The superior pharyngeal constrictor extends from above with four portions, namely: from

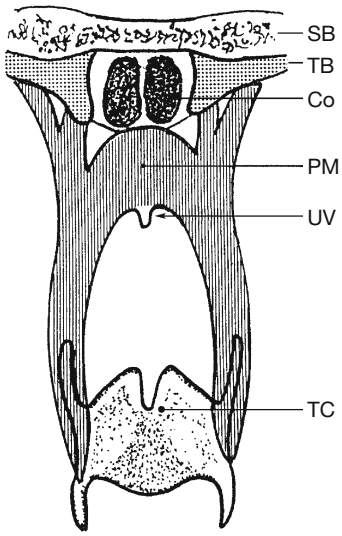

Fig. 1.15. The palatopharyngeal muscle seen from posteriorly. *SB*, skull base; *TC*, tubal cartilage; *CO*, choanae; *SP*, soft palate; *UV*, uvula; *TC*, thyroid cartilage

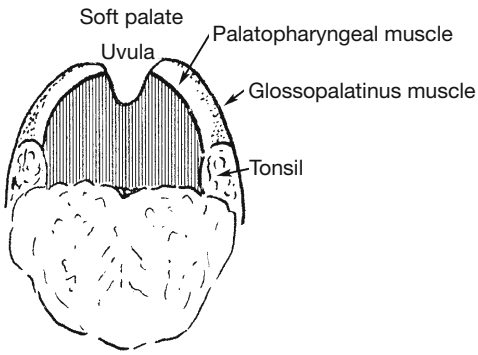

Fig. 1.16. The pharyngeal arches seen from anteriorly. The location of the palatopharyngeal and glossopharyngeal muscles are indicated

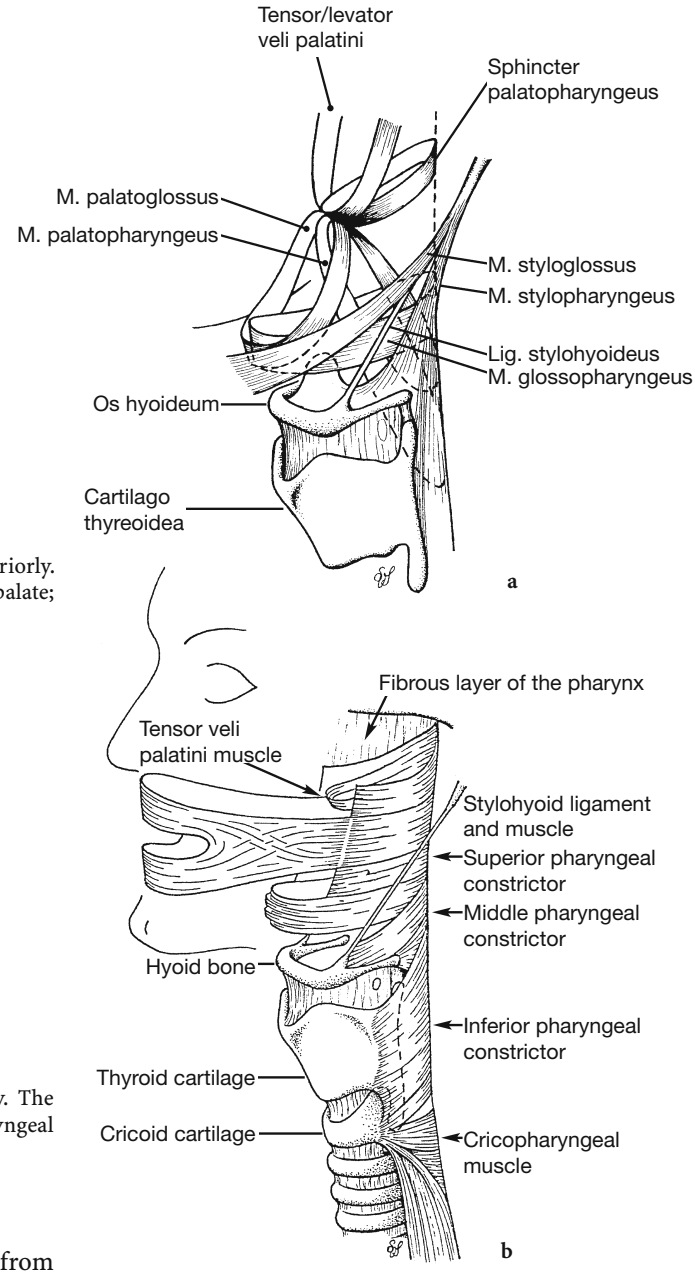

Fig. 1.17a,b. The pharyngeal musculature seen from behind. **a** Palatopharyngeal muscles and elevator of the pharynx. **b** Constrictor muscles (Drawing by Sigurdur V. Sigurjonsson)

the pterygoideus process of the sphenoid bone, from the pterygomandibular raphe, from the mylohyoid line on the mandible and also from the transverse musculature of the tongue. These muscle bundles join and extend posteriorly. They make up the wall of the pharynx and meet in the midline dorsally in the pharyngeal raphe (Figs. 1.17, 1.18).

The middle pharyngeal constrictor extends from the hyoid processes and from the stylohyoid ligament. This ligament runs from the styloid process in the skull base to the minor processes of the hyoid bone. It then extends as a plate posteriorly and superiorly joining the muscles from the other side in the posterior midline in the pharyngeal raphe (Figs. 1.18, 1.19).

The inferior pharyngeal constrictor extends from the cricoid cartilage, from the thyroid cartilage and also from the lateral thyrohyoid ligament (Figs. 1.17–1.20). This muscle extends somewhat superiorly and posteriorly surrounding the pharynx and joining the muscle from the other side in a pharyngeal raphe in the posterior midline. Inferiorly the pharyngeal constrictors form a superiorly convex arch.

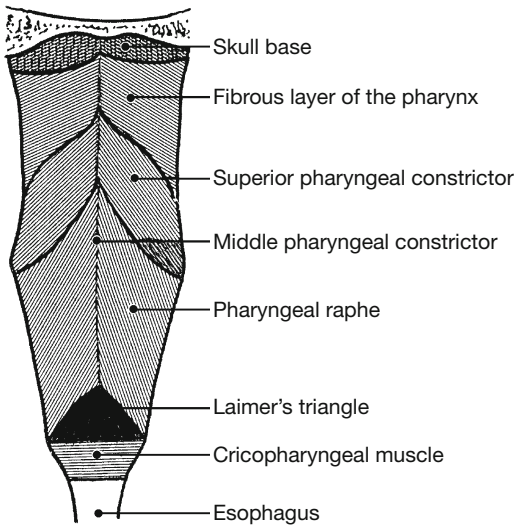

Fig. 1.18. The pharynx seen from the left (Drawing by Sigurdur V. Sigurjonsson)

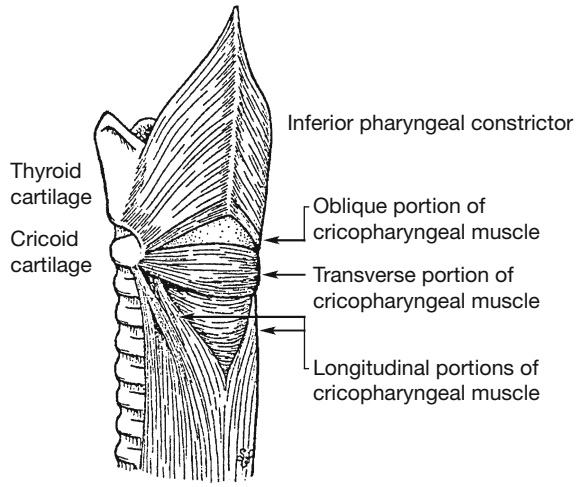

Fig. 1.20. The cricopharyngeal muscle seen from the posterior and left [Drawing by Sigurdur V. Sigurjonsson. From Ekberg and Nylander (1982)]

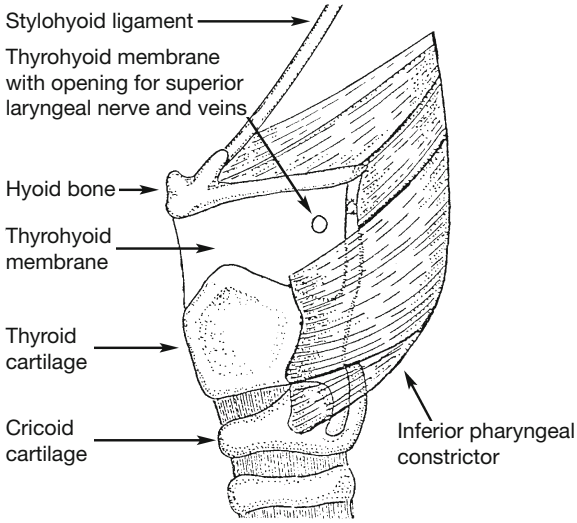

Fig. 1.19. The hyoid bone, thyroid and cricoid cartilage with muscles and membranes seen from the left

There are several muscles that elevate the pharynx. The stylopharyngeal muscle extends from the styloid process and its surroundings at the skull base and extends inferiorly and anteriorly in a gap between the superior and middle pharyngeal constrictors. It partly joins with the contralateral muscles and extends inferiorly to insert on the edges of the epiglottis and also on the posterior margin of the thyroid cartilage (Figs. 1.21, 1.22).

The palatopharyngeal muscle is the biggest of the elevators. It inserts on the posterior border of the hard palate and the palatine aponeurosis and on the pterygoid process. It extends inferiorly and inserts on the back of the thyroid cartilage and also within the constrictor musculature (Fig. 1.15).

1.2.2.4 The Pharyngoesophageal Segment

The pharyngeal constrictors make up the muscle wall of the pharynx almost from the skull base and down into the esophagus. Inferiorly to the constrictors there is one more muscle, namely the cricopharyngeal muscle (Zaino et al. 1970) (Fig. 1.20). This muscle is made up of an oblique portion, a transverse portion (which makes up the bulk of the muscle) and a longitudinal portion of muscle bundles inferiorly. The oblique part extends obliquely, superiorly and posteriorly from the lateral of the cricoid cartilage. It is close to the inferior constrictor. Like the latter muscle it is usually considered that the oblique muscles connect in the pharyngeal raphe. This portion of the cricopharyngeal muscle is anatomically and functionally the inferior (small) portion of the pharyngeal constrictors. The transverse or semicircular portion extends posteriorly from the posterior and lateral part of the cricoid cartilage. Where the two muscles merge in the posterior midline there is *no* fibrous raphe. The two longitudinal muscles, also called esophageal elevators, extend from the inferior portion of the cricoid cartilage and extend

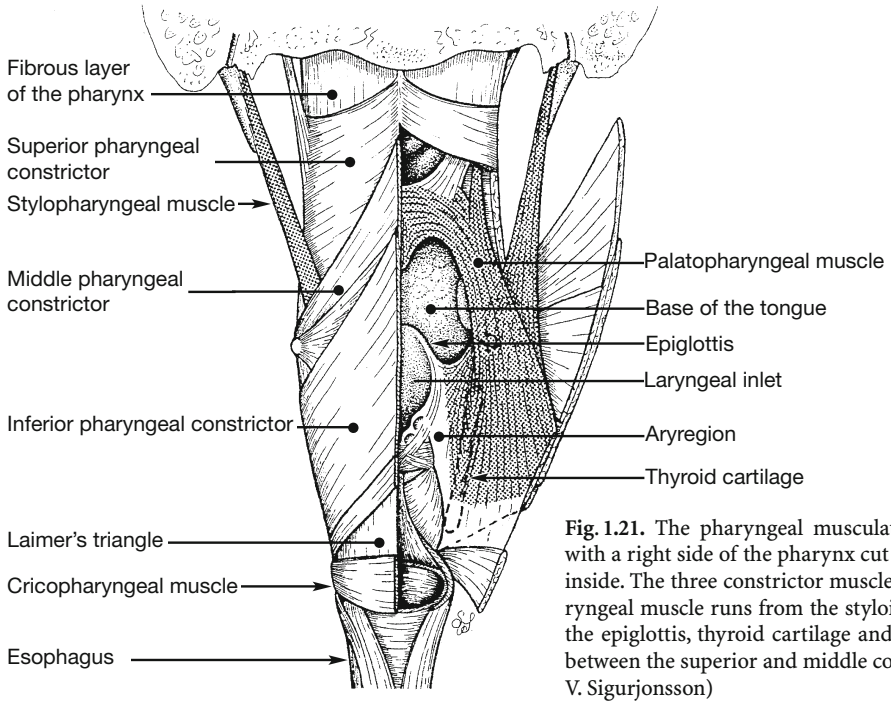

Fig. 1.21. The pharyngeal musculature seen from posteriorly and with a right side of the pharynx cut open so that it can be seen from inside. The three constrictor muscles are overlapping. The stylopharyngeal muscle runs from the styloid process inferiorly to insert on the epiglottis, thyroid cartilage and pharyngeal wall through a gap between the superior and middle constrictors (Drawing by Sigurdur V. Sigurjonsson)

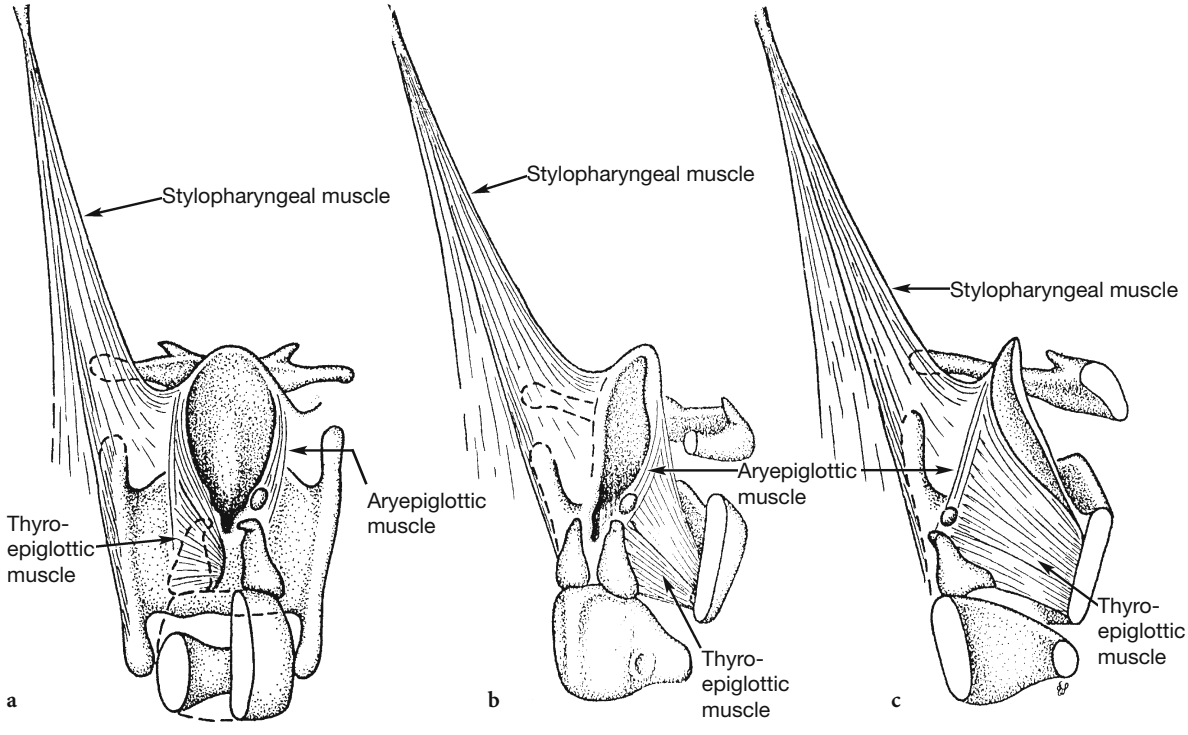

Fig. 1.22a–c. The stylopharyngeal muscle and epiglottic musculature seen from posteriorly (**a**), from posteriorly and right (**b**) and from the right side (**c**) [Drawing by Sigurdur V. Sigurjonsson. Ekberg and Sigurjonsson (1982)]

on each side of the esophagus where they join the longitudinal musculature of the esophagus which in turns comes from the median part of the lamina of the cricoid cartilage. Normally the inferior constrictor muscle overlaps the cricopharyngeal muscle, which in turn overlaps the circular muscle of the esophagus (Ekberg and Lindström 1987). However, between the oblique and transverse part of the cricopharyngeal muscles there is a small triangular gap which is a weak point called Killian's opening or Laimer's triangle. It is through this weak area that the Zenker diverticulum extends. Laterally, there is a similar weak point inferior to the transverse portion and above the insertion of the longitudinal portion of the cricoid muscles. Through this gap the Killian-Jamieson's diverticula extend (Jamieson 1934).

1.2.3 The Larynx

During swallowing, the larynx acts like a valve that closes off the airways from the foodway. The closure of the larynx is achieved by the following mechanisms: The tilting down of the epiglottis is to be achieved in a clear-cut two-step fashion. The first movement is from the upright resting position of the epiglottis to a transversal position. This movement can be explained as consequential to the elevation of the hyoid bone and the approximation between the thyroid cartilage and the hyoid bone. This movement of the epiglottis is thereby the result of contraction of the muscles that elevates the hyoid bone, namely: m. stylohyoideus, m. digastricus, m. mylohyoideus and m. geniohyoideus. In addition, the thyrohyoid muscle approximates the hyoid bone and the thyroid cartilage. The epiglottis is laterally fixed by the pharyngo-epiglottic plicae and, during laryngeal elevation and thyroid approximation to the hyoid bone, is tilted to the transversal position with these plicae as turning points. The second movement of the epiglottis has been attributed either to the passing bolus which should push the movable lip of the epiglottis further down into the esophageal inlet or to the peristaltic contraction in the pharyngeal constrictor musculature. It is more probable that the second movement of the epiglottis is accomplished by one of the muscles that inserts on the epiglottis. These muscles are the stylopharyngeal, thyroepiglottic and aryepiglottic. None of these muscles has such a direction that it is able to tilt down the epiglottis from its upright resting position. However, when the epiglottis has attained a transversal position the conditions may have changed. Still, the stylopharyngeal muscle cannot possibly bring about the second movement, but it is more likely that a contraction in this muscle results in a tilting back of the epiglottis to the upright position. It is possible that the aryepiglottic muscle may be able to pull the epiglottis downwards against the "ary" region, but never as far down as into the esophageal inlet. When these two muscles have been excluded, the thyroepiglottic muscle remains as an able candidate to accomplish the tilting down of the epiglottis. With the epiglottis in the transversal position this muscle has a favourable direction in relation to the epiglottis. A contraction of the thyroepiglottic muscle is therefore very likely to pull the epiglottis down over the ary region. Furthermore, it will change the form of the epiglottis from a downward convex to an upward convex one. A contraction of the aryepiglottic muscle will in this new position of the epiglottis with its tip in the esophageal inlet tighten the laryngeal inlet in the same manner as the string in a tobacco pouch. It is possible to distinguish two different steps in the closure of the vestibule, both of which are clearly separated from the closure of the rima glottidis. In the first step the supraglottic space of the vestibule is closed by the apposition of the lateral walls. This closure of the supraglottic space is caused by contraction and thickening of the superior portion of the thyroarytenoid muscle. The compressed supraglottic space has an orientation in the sagittal plane.

In the second step the closure of the vestibule is effected by a compression of the subepiglottic space from below. This is caused by the posterior aspect of the epiglottis with its superimposed fat cushion that gradually is pressed against the prominence of the ary region. The compressed subepiglottic space has an orientation nearly in the horizontal plane with its anterior part more caudally than the posterior. The tilting down of the epiglottis is probably due to a contraction of the thyroepiglottic muscles. A backward bulging of the superior-anterior wall of the vestibule is achieved by a folding of the median soft tissue linking the thyroid cartilage to the hyoid bone. This tissue comprises the epiglottic cartilage, the pre-epiglottic fat cushion and its bounding ligaments, namely, the thyroepiglottic, the median thyrohyoid and the hyoepiglottic ligaments. In analogy with other folds in this region the above structures have been designated "the median thyrohyoid fold" (Fink 1976).

The described sequence of events in the closure of the vestibule by a compression from below – the supraglottic followed by the subepiglottic space – is

important as it implies a peristaltic-like mechanism that can clear the vestibule from bolus material. After a swallowing act the vestibule is free from foreign particles when it opens again.

The thyroepiglottic muscle and the aryepiglottic muscles pull the epiglottis downwards over the laryngeal inlet (Fig. 1.22). The aryepiglottic muscle runs within the aryepiglottic folds from the ary cartilage in a superior and anterior direction and inserts on the lateral border of the epiglottis (Fig. 1.22). Within the larynx there are several muscles, namely the dorsal cricoarytenoid muscles, the lateral cricoarytenoid muscles and the arytenoid muscle (Figs. 1.23, 1.24). The dorsal cricoarytenoid muscle runs from the posterior surface of the cricoid cartilage superiorly and laterally to insert on the lateral and inferior corner of the arytenoid cartilage. The lateral arytenoid muscle runs from the lateral on the cricoid cartilage superiorly and posteriorly to insert in the same area as the prior described muscle. The arytenoid muscle runs between the two arytenoid cartilages and has a pars recta and also a pars obliqua (Fig. 1.24). The thyroarytenoid muscle runs from the inside of the lamina of the thyroid cartilage and runs dorsally and laterally to insert on the arytenoid cartilage (Fig. 1.25a). It creates a muscle plate that laterally covers the larynx and the inlet to the larynx. The inferior portion is more bulky and it is made up of a lateral part and a vocal part. This latter is often called musculus vocalis within the vocal folds. The somewhat weaker and superior portion of the thyroarytenoid muscle is sometimes called the musculus ventricularis because it forms the plica ventricularis. The thyroarytenoid muscle closes the rima glottidis and at the same time compresses the inferior portion of the laryngeal vestibule which we call the supraglottic space.

The cricothyroid muscle is a strong muscle that runs between the cricoid and thyroid cartilages. The pars recta of this muscle runs superiorly and posteriorly from the cricoid cartilage and inserts on the thyroid cartilage. The pars obliqua of the muscle runs from the cricoid cartilage superiorly and posteriorly to insert on the inferior cornu of the thyroid cartilage (Fig. 1.25b).

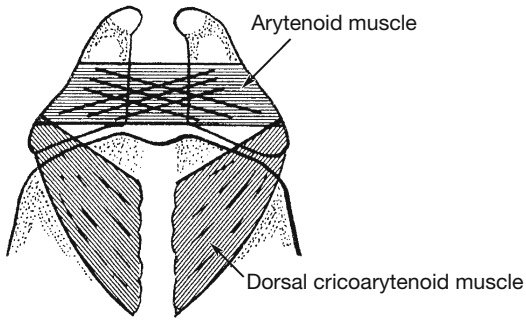

Fig. 1.24. The cranial portion of the cricoid cartilage, the arytenoid cartilage and muscles seen from posteriorly

1.2.4 The Mucosal Surface

Above has been described a framework of bones, cartilages, ligaments, and muscles, constituting the oral cavity, larynx and pharynx. Inside this framework is the mucous membrane (Fig. 1.26, 1.27).

The posterior part of the tongue reaches all the way to the vallecula. This corresponds to the level of the hyoid bone. Here is a pocket on each side of the midline, the vallecula. Posteriorly and laterally the valleculae are bordered by a mucosal fold above the stylopharyngeal muscle. This fold is called the pharyngoepiglottic fold. The two valleculae are separated in the midline by a mucosal fold, the median glossoepiglottic fold (Figs. 1.26, 1.27). The tongue base and valleculae contain a rich network of lymphatic tissue. The vallecula may also contain vessels in the submu-

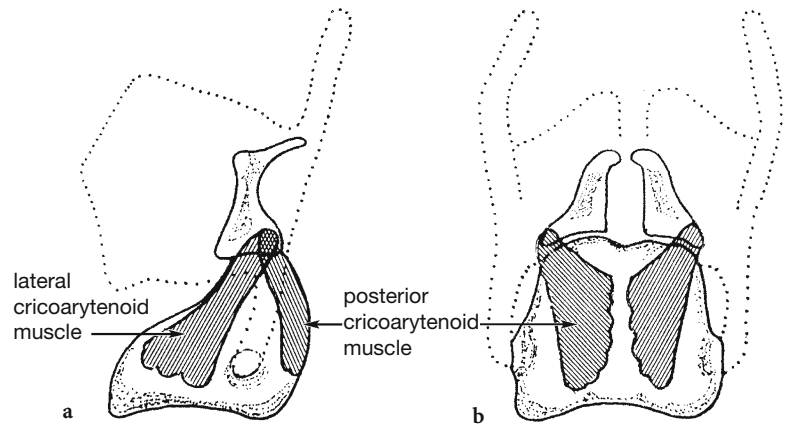

Fig. 1.23a,b. The cricoid cartilage, arytenoid cartilage and muscles seen from the left (**a**) and from posteriorly (**b**)

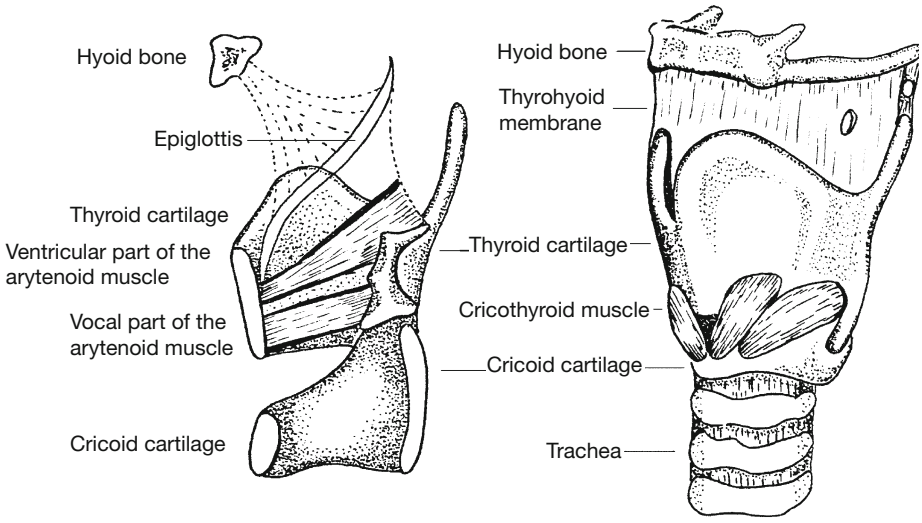

Fig. 1.25. The larynx cut open in the midline and seen from the left side. The larynx seen from anteriorly and left

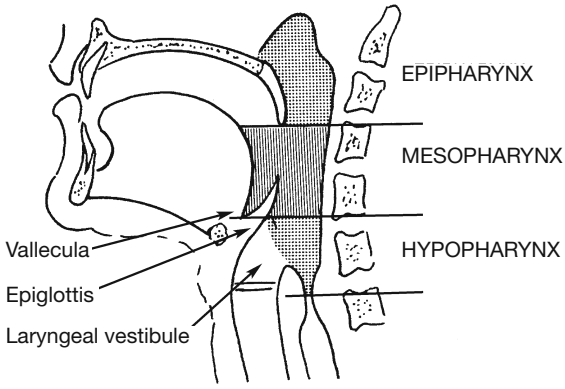

Fig. 1.26. Schematic drawing of the pharynx seen from the left

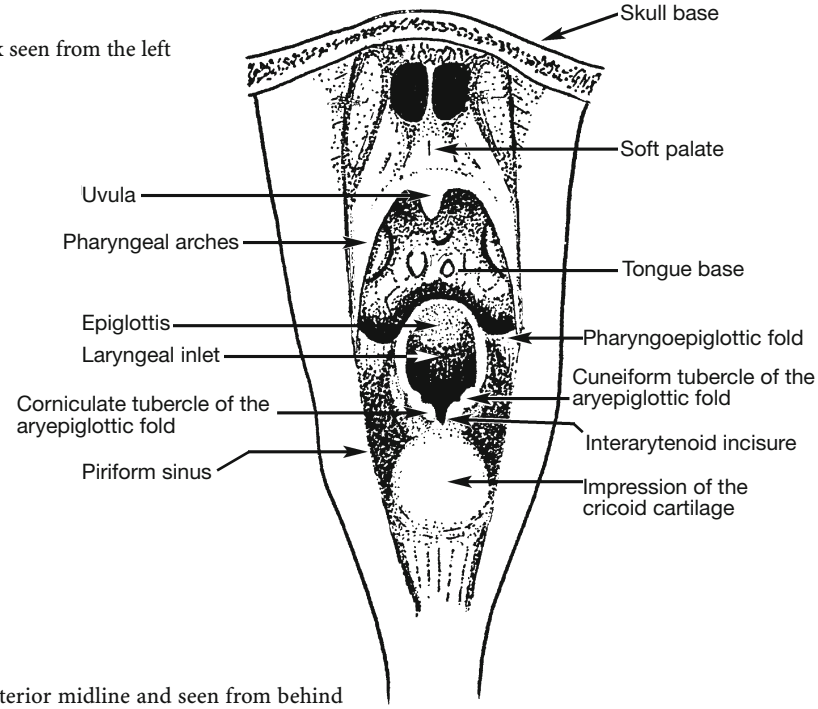

Fig. 1.27. The pharynx cut open in the posterior midline and seen from behind

cosa that causes a web-like appearance (Ekberg et al 1986). Further inferiorly (Fig. 1.27) there is a fold reaching from the lateral border of the epiglottis to the ary region. The folds surround the inlet of the laryngeal vestibule. This is the aryepiglottic fold which harbours the aryepiglottic muscle. There are two small protuberances caudally/inferiorly due to the cuneiform tuberculum superiorly and corniculate tubercle inferiorly. Between the two corniculate tubercles there is a cleft called the interarytenoid incisure. The aryepiglottic fold is made up of the aryepiglottic muscle posteriorly and anteriorly of the thyroepiglottic muscle. The lamina of the cricoid cartilage causes an impression of the pharyngeal lumen. On both sides of these impressions there are two recesses called the piriform sinuses.

1.3 Anatomy of the Esophagus

The esophagus can be divided into different parts according to the surrounding anatomical structures (Fig. 1.28). The superior part, the pharyngoesophageal segment (PES, functional term), also called the upper esophageal segment (UES, anatomical term), corresponds to the cricopharyngeal muscle and surrounding pharynx and cervical esophagus. This is also called introitus esophagi or Killian's mouth. From here to the impression of the aorta is the paratracheal esophagus (Fig. 1.28). This is located close to the membranous part of the trachea. The aorta makes a short impression from the left into the aortic lumen. Inferiorly to this and above the left main bronchus is the aortobronchial portion which is a short, relatively wide segment. The left main bronchus makes a short impression in the esophagus from the left. The cardial portion is that segment of the esophagus which is located close to the left atrium of the heart. A schematic drawing of the gastro-esophageal region is seen in Fig. 1.29.

The esophagus is made up of three layers, the mucosa, the submucosa and the muscularis (Fig. 1.30). The mucosa is made of squamous cell epithelium. Under the epithelium there is a submucosal layer of musculature as everywhere else in the alimentary canal. The mucosa also contains glands and vessels. The mucosa has a tendency to create longitudinal mucosal folds.

The esophagus has two layers of muscles, an inner circular and an outer longitudinal muscle layer. The longitudinal muscles insert on the posterior aspect of the lamina of the cricoid cartilage. The upper third of the esophagus is made up of striated musculature while the lower two thirds are smooth muscles. The transitional zone, however, has a varying position. The circular muscle layer is thinner cranially and increases in thickness distally. Between the two muscle layers there is a multitude of neurons in a plexus formation (Auerbach's plexus). In this there are both sympathetic and parasympathetic nerves. There is a close proximity between the vagus nerve and the esophagus, especially inferiorly.

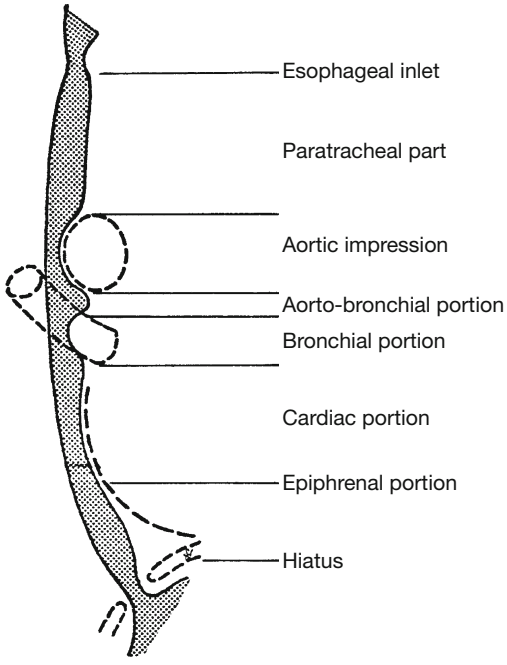

Fig. 1.28. Schematic drawing of the esophagus illustrating the different parts of the esophagus

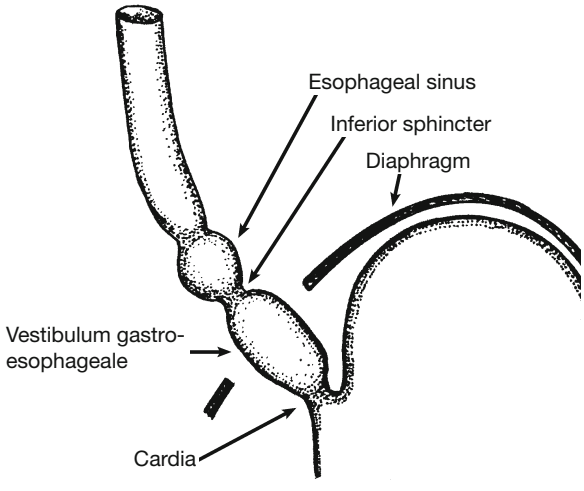

Fig. 1.29. The gastro-esophageal region

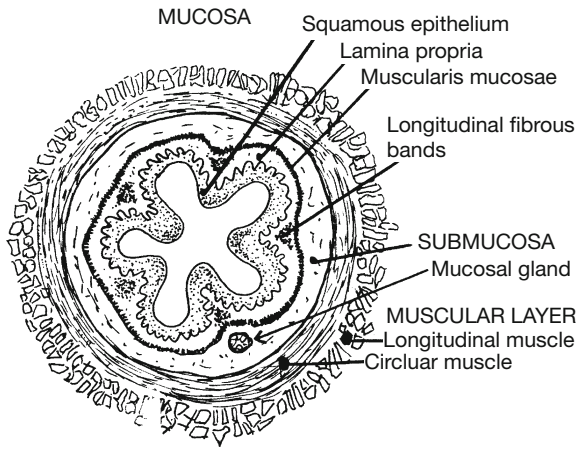

Fig. 1.30. Cross section of the esophagus

1.4 Neuroanatomy and Physiology of Swallowing

There are several reviews on the neuroanatomy and neurophysiology of swallowing, the most contemporary by Miller (1999). Several of the cranial nerves are involved in the control of swallowing (Perlman and Christensen 1997). Oral sensation is transmitted in the trigeminal nerve. Efferent information in the trigeminal nerve goes to the mylohyoid muscle, anterior belly of the digastric, and the four muscles of mastication: the masseter, temporalis and pterygoid muscles.

Taste sensation is mediated in the facial nerve. Efferent control from the facial nerve goes to the salivary glands and to muscles of facial expression, the stylohyoid and platysma, as well as the posterior belly of the digastric.

The glossopharyngeal nerve conveys taste information from the posterior tongue. It also conveys sensation from the pharynx. It innervates only the stylopharyngeal muscle efferently.

The vagus is the most important nerve for swallowing. It innervates the pharyngeal and laryngeal mucosa. The recurrent laryngeal nerve conveys sensation from below the vocal folds and also the esophagus. Efferent control in the vagus nerve comes from the nucleus ambiguus (striated muscle) and the nucleus dorsalis nervi vagi (smooth muscles and glands).

The hypoglossal nerve provides efferent control of all the intrinsic and some of the extrinsic muscles of the tongue.

The locations of the central swallowing pathways include several cortical and subcortical regions. One such area is located immediately in front of the precentral sulcus cortex. Stimulation in this area evokes mastication followed by swallowing. It is likely that the cortical and subcortical areas merely modify swallowing as pharyngeal and esophageal swallowing can be evoked also in the absence of these areas. This indicates that the brainstem is the primary swallowing area.

Afferent information from the oral cavity and pharynx is mediated via the vagus nerve and others to the nucleus tractus solitarius (NTS) in the brainstem. Close to the NTS is an afferent swallowing centre that interprets the information. If it is found appropriate for swallowing, information goes to a swallowing centre close to the nucleus ambiguus. From that swallowing centre control of the pharynx is managed. Information also goes to a dorsal swallowing centre close to the nucleus dorsalis nervi vagi. The oral stage of swallowing is completely voluntary while the pharyngeal stage of swallowing is automatic. This automatism means that there is a none-or-all situation. Once the pharyngeal swallow is elicited it is always completed. It is not modified during the pharyngeal swallowing process and it cannot be interrupted. Swallowing has priority over other activities controlled from the nucleus ambiguus such as breathing, speech and positioning. The esophageal stage of swallowing is autonomic which means that it may occur also without control from the brainstem. It is also self-regulatory, i.e. a second swallow interrupts the first, and a secondary peristaltic wave can be elicited. This is achieved by the enteric nervous system.

The oral stage of swallowing includes ingestion which is a complex act. It also involves blending, mixing and mincing of ingested material. When the ingested material is found to be appropriate for swallowing (by analysing information from the nucleus tractus solitarii), the tongue usually scoops up a suitable amount of ingested material, which is from now on called a bolus, on to the top of the tongue. From there it is propelled by a sweeping movement of the tongue into the pharynx. The pharyngeal stage of swallowing includes sealing off of the nasopharynx with the soft palate opposing the posterior pharyngeal wall and also the closing of the airways by elevation and closure of the larynx and tilting down of the epiglottis. Opening of the PES is also mandatory.

The pharyngeal constrictors achieve the final rinsing of the pharynx. An important item is the elevation of the pharynx and larynx. When the bolus reaches the upper part of the esophagus peristaltic activity occurs. This means that esophageal tonicity is abolished and the bolus is propelled downwards by a combination of gravity and contraction in the circular musculature. When this occurs in connection

to pharyngeal swallowing this is called primary peristalsis. If it occurs by local distension, for instance by retained material or regurgitated/reflux material, it is called secondary peristalsis. If contraction is nonpropulsive it is called simultaneous contraction. In the elderly patient this has also been called tertiary contraction.

References

Bosma JF (ed) (1973) Fourth symposium on oral sensation and perception: Development in the fetus and infant. DHEW Publication No. (NIH) 73-546 Bethesda, Maryland

Bosma JF (ed) (1976) Symposium on development of the basicranium. DHEW Publication No. (NIH) 76-989, Bethesda, Maryland

Ekberg O, Lindström C (1987) The upper esophageal sphincter area. Acta Radiol 28:173–176

Ekberg O, Birch-Iensen M, Lindström C (1986) Mucosal folds in the valleculae. Dysphagia 1:68–72

Ekberg O, Nylander G (1982) Dysfunction of the cricopharyngeal muscle. A cineradiographic study of patients with dysphagia. Radiology 143:481–486

Ekberg O, Sigurjonsson SV (1982) Movement of the epiglottis during deglutition. A cineradiographic study. Gastrointest Radiol 7:101–107

Fink BR (1976) The median thyrohyoid "fold": a nomenclature suggestion. J Anat 122:697–699

Fink BR, Demarest RJ (1978) Laryngeal biomechanics. Harvard University Press, Cambridge, MA

Jamieson JB (1934) Illustrations of regional anatomy. Section 2:44. E&S Livingstone Ltd, Edinburgh

Miller AJ (1999) The neuroscientific principles of swallowing and dysphagia. Singular Publishing Group, San Diego.

Perlman AL, Christensen J (1997) Topography and functional anatomy of the swallowing structures. In: Perlman AL, Schulze-Delrieu K (eds) Deglutition and its disorders. Anatomy, physiology, clinical diagnosis, and management. Singular Publishing Group, Inc., San Diego, pp 15–42

Williams PL, Warwich R, Dyson M, Bannister LH (eds) (1989) Gray´s anatomy, 37th edn. Churchill Livingstone, Edinburgh

Zaino C, Jacobson HG, Lepow H, Ozturk CH (1970) The pharyngoesophageal spincter. Charles C. Thomas Publ, Springfield, IL

2 Evaluation of Symptoms

Doris-Maria Denk

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2.1. Introduction

Symptoms of pharyngeal/esophageal diseases are related to swallowing function, such as dysphagia, odynophagia, globus sensation or heartburn. Patients often do not differentiate between these symptoms and report "swallowing problems". The crossing of airway and digestive tract in the hypopharynx is a critical region for swallowing and respiration: if protection of the airway during swallowing is not secured, aspiration occurs. Moreover, the pharynx is not only part of the upper digestive tract, but also of the vocal tract and therefore influences resonance and articulation.

Swallowing belongs to the most frequent activities in the human body: the human being swallows between 580–2000 times a day (Logemann 1998; Garliner 1974). However, swallowing is not only a vital primary function to ensure adequate nutrition and hydration, but also decisively contributes to quality of life and social integration. Dysphagia may lead to malnutrition and, in the case of aspiration, to potentially life-threatening pulmonary complications, such as aspiration pneumonia. Furthermore, life-quality of dysphagic patients is impaired (Ekberg et al. 2002). Dysphagia represents a frequent and severe medical problem: in acute care hospitals, 13%–14% of patients are believed to suffer from dysphagia, in nursing homes the percentage of dysphagic patients reaches up to 50% (Logemann 1995). Moreover, among patients aged over 65 years, aspiration pneumonia is the fourth most frequent cause of death (Sasaki 1991). Every year, about 50,000 US Americans die from pulmonary complications of aspiration (Jones and Donner 1991). Therefore, in modern, function-orientated medicine, the management of the dysphagic patient has become of great clinical importance and a focus of scientific interest.

Dysphagia and other swallowing complaints necessitate a thorough diagnostic procedure. Only the knowledge of the underlying cause and of the individual swallowing pathophysiology enables appropriate treatment of the patient.

2.2. Terminology of Dysphagia

The symptom *dysphagia* is defined as disturbance of the intake or transport of food from the mouth to the stomach. Furthermore, it includes behavioural, sensory and motor disorders in preparation for the swallow, e.g. disorders of cognitive awareness, visual and olfactory recognition of food and of the physiologic responses to smell and presence of food (Leopold and Kagel 1996). In the case of *oropharyngeal dysphagia*, the oral preparatory, oral and/or pharyngeal phases of swallowing are afflicted. If the esophageal phase is disturbed, *esophageal dysphagia* is present. Both types of dysphagia may influence each other, therefore dysphagia makes the comprehensive evalu-

D.-M. Denk, MD
Professor, Department of Otorhinolaryngology, Section of Phoniatrics, University of Vienna, Vienna Medical School, Währinger Gürtel 18–20, 1090 Vienna, Austria

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ation of the aerodigestive tract from the oral cavity to the stomach necessary.

2.2.1. Components of the Impaired Swallow

To address the pathophysiological aspects, dysphagia has to be regarded as a syndrome. The most important dysphagia-related symptom is *aspiration*, which is defined as the entry of saliva, food or gastric secretion into the airway under the level of the vocal folds. Other components of dysphagia are drooling, leaking, nasal penetration, laryngeal penetration, retention or pharyngeal regurgitation. The swallowing pathophysiology is analysed in relation to the phases of swallowing.

Drooling describes complaints of oral spill, i.e. the falling of food, liquid or saliva from the mouth anteriorly when lip closure is incomplete. *Leaking* is defined as premature loss of the bolus over the tongue base into the pharynx before the swallowing reflex is triggered. Consequently, the risk of aspiration occurs. A *delayed triggering of the swallowing reflex* occurs in neurological diseases (e.g. stroke) or after extensive surgical resection of the trigger points for the pharyngeal swallow.

Retention (pooling) of saliva or food may be localized in the oral cavity, valleculae or hypopharynx. Retentions in the anterior or lateral sulcus are due to reduced muscle tone in the labial or buccal musculature. Disturbed lingual function may result in retentions on the floor of the mouth and the valleculae. Weakness, paresis or scarring of pharyngeal muscles give rise to pharyngeal retentions.

Nasal penetration (regurgitation) describes the entry of food into the nose and may be caused by incomplete velopharyngeal closure or pharyngeal/esophageal stop of the bolus passage with subsequent overflow into the nasal cavity. In case of *laryngeal penetration*, food or saliva reaches the larynx to the level of the vocal folds.

Pharyngeal regurgitation is characterized by (parts of) the already swallowed bolus flowing back into the pharynx due to a Zenker's diverticulum or a disturbed esophageal bolus transport.

2.2.2. Dysphagia and Other Swallowing Complaints

Dysphagia has to be distinguished from other swallowing complaints, such as globus sensation or odynophagia. *Odynophagia* describes the painful swallow, as occurs in inflammatory or tumorous diseases of the upper aerodigestive tract (e.g. acute tonsillitis, peritonsillar abscess, epiglottitis, hypopharyngeal carcinoma, etc.).

Globus sensation (*globus pharyngis*) is a feeling of a lump or fullness in the throat and discomfort when swallowing saliva. In contrast to dysphagia, swallowing of food is not disturbed. The symptom mainly occurs during swallowing of saliva and decreases or vanishes while swallowing food. In many patients, an underlying cause can be found, e.g. gastroesophageal reflux disease, esophageal motility disorders, hypertensive upper esophageal sphincter (cricopharyngeal achalasia), thyroid gland disease, cervical spine syndrome or hyperfunctional voice disorder. Therefore, the obsolete term "globus hystericus" should not be used any more. Only if an exact morphological and functional analysis of larynx, pharynx, esophagus and neck does not show any medical entity, a psychogenic aetiology can be suspected. Above all, globus sensation and dysphagia may occur in combination.

2.3. Aspiration

The antero- or retrograde entry of saliva, food or gastric secretion into the airway under the level of the vocal folds is defined as *aspiration* (Fig. 2.1). To reveal or exclude aspiration is the main goal of the diagnostic procedure in dysphagic patients. The

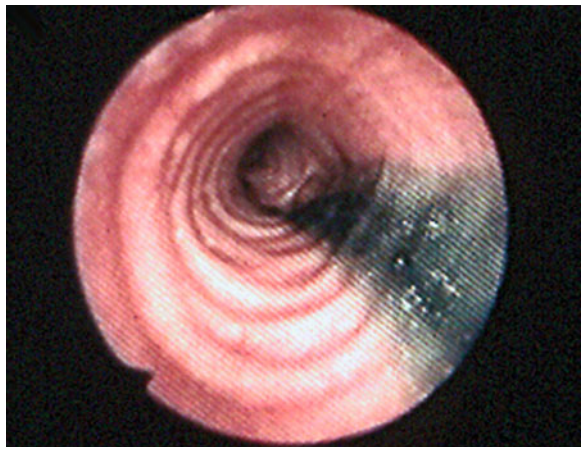


Fig. 2.1. Aspiration (videoendoscopic view): blue coloured aspirated food in the trachea [from Bigenzahn and Denk (1999)]

presence/absence of aspiration determines further patient management. In the case of absent or reduced cough reflex, aspiration does not induce cough, but remains "silent" (*silent aspiration*) and is not immediately noticed. About 40% of aspirating patients are so-called silent aspirators.

Aspiration can be classified in relation to the triggering of the swallow reflex. It may occur before (predeglutitive), during (intradeglutitive), after the swallow (postdeglutitive) or in combined forms (Logemann 1983). *Aspiration before the swallow* may be present when the triggering of the swallow reflex is absent or disturbed, e.g. after stroke. Incomplete laryngeal closure and/or reduced laryngeal elevation may give rise to *aspiration during the swallow*, as is the case, for example, in vocal fold paralysis or in laryngeal defects after partial laryngectomy. Reduced pharyngeal peristalsis, reduced laryngeal elevation and disturbed opening of the pharyngo-esophageal sphincter possibly results in *aspiration after the swallow* (Fig. 2.2), e.g. in fibrosis with "frozen" (immobile) larynx after radiation therapy or cricopharyngeal achalasia after stroke.

The severity of aspiration is not only influenced by the amount and type of the aspirated material, but also by the presence of cough reflex and the possibility of voluntary coughing and throat clearing. Several severity scales are used for the grading of aspiration (Table 2.1). The clinical aspiration scale (Miller and Eliachar 1994) considers possible pulmonary consequences. In the videoendoscopic aspiration scale (Schröter-Morasch 1996) attention is paid to the cough reflex and voluntary coughing. The videofluoroscopic aspiration scale (Hannig et al. 1995) is based on the amount of aspirated material and the presence/absence of the cough reflex.

Table 2.1. Aspiration scales

Clinical Scale (Miller and Eliachar 1994)	
I:	Incidental aspiration without complications
II:	Intermittent aspiration of liquids; saliva and solid boluses can be swallowed
III:	No oral feeding possible, intermittent pneumonias
IV:	Life-threatening aspiration; chronic pneumonia/hypoxia
Videoendoscopic Scale (Schröter-Morasch 1996)	
I:	Incidental aspiration, intact cough reflex
II:	Incidental aspiration, no cough reflex, voluntary coughing possible or Permanent aspiration, intact cough reflex
III:	Permanent aspiration, no cough reflex, voluntary coughing possible
IV:	Permanent aspiration, no cough reflex, no voluntary coughing
Videofluoroscopic Scale (Hannig et al. 1995)	
I:	Aspiration of material that has penetrated into the laryngeal vestibule or ventricle, intact cough reflex
II:	Constant aspiration of less than 10% of the bolus, intact cough reflex
III:	Constant aspiration of less than 10 % of the bolus, reduced cough reflex or Constant aspiration of more than 10% of the bolus, intact cough reflex
IV:	Constant aspiration of more than 10% of the bolus, reduced cough reflex

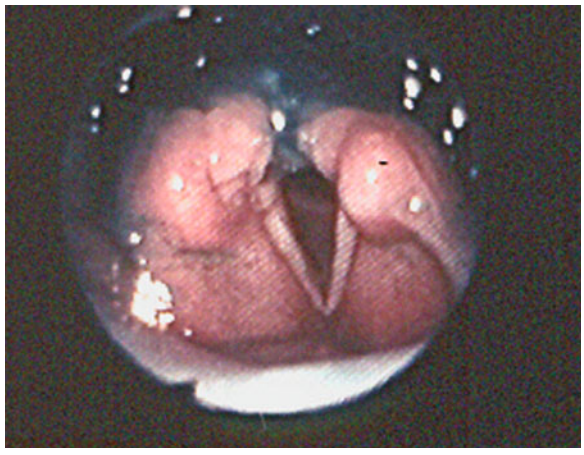


Fig.2.2. Aspiration after the swallow (videoendoscopic view): overflow aspiration due to retentions in the hypopharynx [from Bigenzahn and Denk (1999)]

The individual tolerance of aspiration varies widely. Some patients tolerate aspiration of more than 10% of the bolus, whereas other patients develop aspiration pneumonia even after aspiration of their saliva. Therefore, not only aspiration, but other additional risk factors play an important role. Langmore et al. (1998) found the following predictors for the development of aspiration pneumonia: dependent for feeding, dependent for oral care, number of decayed teeth, tube feeding, more than one medical diagnosis, number of medications and smoking.

2.4. Aetiology of Dysphagia

The aetiologies of dysphagia may be divided into the following groups:

- Diseases of the upper aerodigestive tract (*peripheral "mechanical" dysphagia*)
- Neurological diseases (*neurogenic dysphagia*)
- *Psychogenic dysphagia*

Only if after thorough diagnostics peripheral or neurogenic dysphagia is excluded, psychogenic factors have to be considered. In some cases, the distinction from eating disorders is difficult.

2.4.1. Mechanical Dysphagia

Diseases of the upper swallowing and respiratory tract or surrounding structures may give rise to dysphagia and aspiration (Table 2.2). The symptom dysphagia necessitates the exclusion of a malignant tumour in the aerodigestive tract. Moreover, not only a tumorous disease of the oral cavity, pharynx or larynx itself, but also the sequelae of therapy – surgical resection, radiation, chemotherapy – can interfere with bolus transfer or airway protection with consecutive dysphagia and aspiration that requires a functional swallowing therapy to regain swallowing function. The different tumour resections in the head and neck are known to create patterns of swallowing disorders, but the same

Table 2.2. Examples of dysphagia aetiologies [modified from Denk and Bigenzahn (1999)]

Type of dysphagia	Aetiology
Mechanical peripheral dysphagia	
Oropharyngeal	Inflammatory diseases
	Malignant tumours in the upper aerodigestive tract
	Sequelae after tumour therapy (surgery, radiation, chemotherapy)
	Diseases/surgery of the cervical spine
	Long-term-intubation
	Cheilognathopalatoschisis
	Tracheo-esophageal fistula
	Diverticula (Zenker's diverticulum)
	Goitre
	Systemic diseases (scleroderma, amyloidosis)
	Graft-versus-host-disease
Esophageal	Obstructive esophageal diseases (peptic, tumorous stenosis)
	Motility disorders (gastro-esophageal reflux disease, Non-propulsive contractions)
Neurogenic dysphagia	
Central nervous system	
	Stroke
	Degenerative processes: amyotrophic lateral sclerosis, Parkinson's disease, Multiple sclerosis
	Cerebral palsy
	Dementia, Alzheimer's disease
	Post-polio syndrome
	Encephalitis
	AIDS
	Posterior fossa tumours
	Head trauma, cervical spine cord injury
	Intoxications
	Drug effects (sedatives, neuroleptics)
	Arnold Chiari malformation
Peripheral nervous system	
	Skull base tumours (chordoma, meningioma)
	Meningitis
	Guillain-Barré syndrome
	Neuropathy (alcoholic, diabetic)
Neuromuscular junction	
	Myasthenia gravis
	Botulism
	Lambert-Eaton syndrome
Muscles	
	Dermatomyositis, polymyositis
	Myopathy (endocrine/metabolic)
	Myotonia, muscular dystrophy
Psychogenic dysphagia	
	Phagophobia

resections need not necessarily result in the same form and degree of dysphagia and aspiration. The extent and localization of the resections carried out are regarded as determining factors for the severity of dysphagia. Beside these local factors, also general factors, e.g. patients' general condition or therapy onset postoperatively, influence the outcome of swallowing rehabilitation (Denk and Kaider 1997).

Not only tumours in the pharynx or esophagus, but also thyroid gland disease or cervical osteophyte compression due to diffuse idiopathic skeletal hyperostosis (DISH) (Marks et al. 1998) may be responsible for obstructive dysphagic symptoms which are typically worse for solid than liquid bolus. Patients suffering from DISH (Fig. 2.3) become especially symptomatic when an additional disease afflicting swallowing function (e.g. stroke) impairs the patient's functional compensatory capability.

2.4.2. Neurogenic Dysphagia

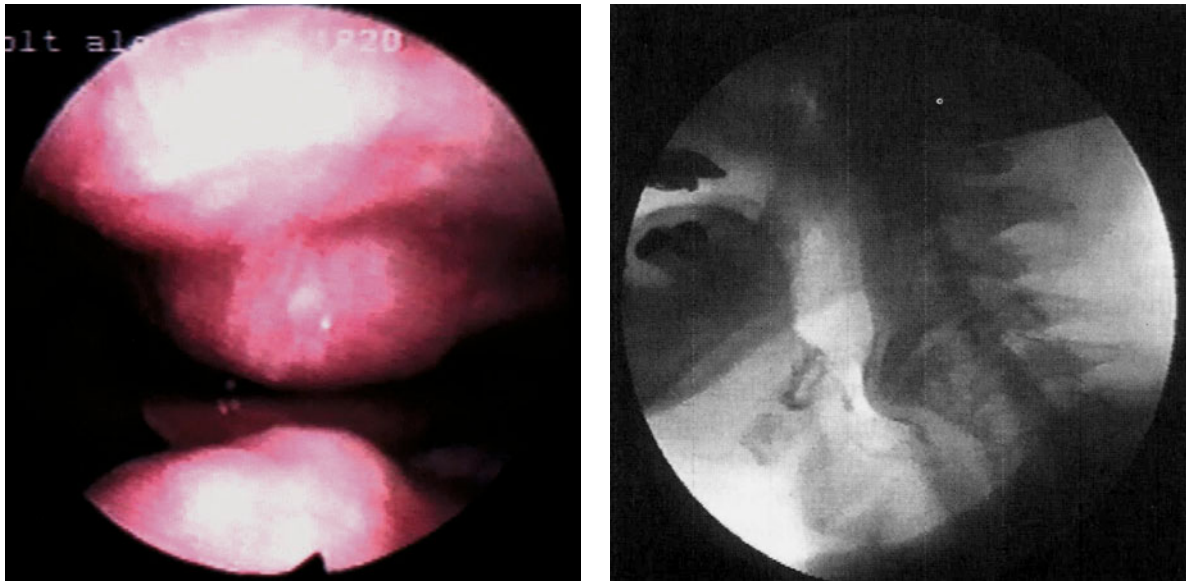
Nearly all neurological diseases have the potential to disturb the four levels of sensomotoric control of the swallow (central, peripheral nervous system, neuromuscular junction, muscles) and may cause dysphagia and aspiration (Table 2.2). For the management of the patients (functional therapy, type of nutrition) it is of utmost importance to distinguish between neurologic lesions with recovery potential (e.g. stroke, head trauma, cervical spine cord injury, etc.) and progressive diseases.

Stroke represents the most frequent cause of dysphagia (25%, Groher and Bukatman 1986). The percentage of dysphagic stroke patients differs with the time from the onset of stroke: in the first 2 weeks after stroke 41%, in the chronic phase 16% of the patients suffer from dysphagia (Kuhlemeier 1994). After all, within the first year after stroke, 20% of patients die from aspiration pneumonia (Brown and Glassenberg 1973).

Beside the swallowing disturbance, neurologic patients possibly show additional symptoms that have to be considered. Disturbances in the motor system bring about impaired posture and head control, and cognitive deficits lead to a lacking awareness of disease. Severely impaired speech and language (e.g. dysarthria, aphasia) impair communication with the patient.

2.5. Clinical Symptoms of the Dysphagic Patient

Aspiration is well known as the most threatening symptom of dysphagia. Due to disturbed laryngeal sensibility and absent cough reflex, aspiration often occurs silently. Therefore the fact that the patient does not cough/choke while eating cannot be regarded



a b

Fig.2.3a,b Diffuse idiopathic skeletal hyperostosis (DISH). **a** Endoscopic view. **b** Radiologic view [from Bigenzahn and Denk (1999)]

as a reliable "clue" symptom to exclude aspiration. *Indirect and direct symptoms of dysphagia/aspiration* (Table 2.3) are possible hints to suspect dysphagia and aspiration. Direct symptoms occur during swallowing of food and liquids, whereas indirect symptoms are not directly associated with the swallow as such, but are due to dysphagia.

Among the *indirect symptoms*, weight loss is regarded as a reliable hint to judge the effects of swallowing impairment, because weight is usually directly related to the nutritional state. Frequent occurrence of fever, coughing, bronchitis or pneumonia may be clinical consequences of aspiration. Changes of voice (dysphonia), speech (dysarthria) and language (aphasia) should not be neglected, as they may be related to neurologic diseases. Moreover, anatomical and functional deficits in the upper aerodigestive tract possibly also lead to dysphonia, altered resonance (e.g. hyperrhinophonia = too much nasal resonance) or impaired articulation (e.g. dysglossia = disturbed articulation due to changes in the peripheral organs of speech). Globus sensation, heartburn and/or non-cardiac chest pain often are present in gastroesophageal reflux disease or esophageal motility disorders. Alterations of taste or mucosal dryness impair swallowing function and the pleasure from oral intake.

Prolonged duration of swallowing, pain (odynophagia) or fear of swallowing belong to the *direct symptoms of dysphagia and aspiration*. Furthermore, changes in posture during oral food intake and changes in eating habits (e.g. avoidance of some food consistency) deserve clinical awareness. Other direct symptoms the patient may report are drooling, nasal regurgitation, choking or coughing during or immediately after the swallow due to aspiration, spitting of food or regurgitation. The feeling of obstruction may occur not only in patients with tumours, strictures, Zenker's diverticulum, webs or cervical osteophytes (DISH), but also in neurologic diseases because of pharyngeal muscle weakness, lack of coordination of the swallow or esophageal motility disorder.

2.6. Diagnostic Procedure

For adequate management of the patient suffering from swallowing complaints a thorough morphological and functional diagnostic procedure is needed to evaluate the swallow from the oral cavity to the stomach and to reveal the aetiology and individual swallow profile. The diagnostic procedure is summarized in Table 2.4. To address the complexity of swallowing disorders, an interdisciplinary approach is necessary. Very often, the dysphagic patient first presents to the otorhinolaryngologist/phoniatrician who – after thorough history taking – performs a videoendoscopy of the upper aerodigestive tract and videoendoscopic swallowing study (fiberoptic endoscopic evaluation of swallowing (FEES), (Langmore et al. 1988; Bastian 1991), and refers the patient to a videofluoroscopic swallowing study. Depending on the patient's needs and findings, fur-

Table 2.3. Symptoms of dysphagia/aspiration [from Schröter-Morasch (1993)]

Indirect symptoms
Weight loss
Frequent occurrence of fever
Coughing
Bronchitis/pneumonia
Changes of voice, articulation/speech and language
Globus sensation
Heartburn
Non-cardiac chest pain
Direct symptoms
Prolonged duration of swallowing
Pain
Fear of swallowing
Changes of posture
Avoidance of particular consistencies
Drooling
Obstruction
Choking, coughing
Spitting of food
Regurgitation

Table 2.4. Diagnostic procedure [from Denk and Bigenzahn (1999)]

History
Basic Diagnostics (compulsory)
● ENT/phoniatric examination with clinical observation and videoendoscopic swallowing study (= flexible endoscopic evaluation of swallowing, FEES)
● Videofluoroscopic swallowing study
● If indicated, esophagoscopy
● In the case of aspiration, chest X-ray
Further diagnostics (optional)
● Esophagoscopy, gastroscopy
● Ultrasound of the oral phases of swallowing Cranial MRI Scintigraphy
● Manometry, pH-metry
● Electrophysiological methods (electromyography)

ther examinations have to be performed and further medical disciplines need to get involved, such as gastroenterology, pulmonology, neurology, surgery, maxillo-facial surgery, etc.

The diagnostic procedure aims at revealing the components of dysphagia, especially proving or excluding aspiration. Moreover, a classification and quantification of aspiration has to be performed as well as a prognostic estimate. Further diagnostic goals are recommendations of therapy and type of feeding, as well as indications for emergency therapies (such as tracheostomy) in the case of intractable aspiration.

2.6.1. Patient History

Patient history provides valuable information that helps to optimize the diagnostic work-up. The patient is asked to characterize his complaints and to describe their beginning, time-characteristics (intermittent or constant occurrence) and influencing factors. However, it is important to know about the influence of bolus consistency on swallowing. In neurologic patients with impaired swallowing reflex and uncoordinated swallow, liquids are more difficult to be swallowed than semisolids or solids, because they cannot be controlled well. Patients with obstructive diseases (tumours, strictures, web) often report sticking of solid food, whereas patients suffering from esophageal motility disorders complain about swallowing problems with both liquids and solid food. To estimate the patient's oral intake, a description of his meals and weight during the last week may be valuable.

Furthermore, information regarding previous diseases and therapies (surgery, radiation), as well as medication, needs to be obtained. The side effects of many drugs can impair swallowing: psychopharmacological drugs possibly interfere with the swallow reflex or induce xerostomia, antipsychotic drugs can cause extrapyramidal symptoms, spasmolytics may weaken muscles. It has to be pointed out that changes of voice, speech and language should be carefully noted. Above all, the examiner should also take into consideration the patient's general condition, his posture and cognitive and emotional state.

2.6.2. Basic Diagnostic Procedure

The ENT examination plays an important "key" role in the diagnostic work-up because it allows a direct morphological and functional analysis of the upper aerodigestive tract. Since clinical observation and palpation of the swallow alone do not meet the diagnostic demands, a videoendoscopic swallowing study, (FEES, fiberoptic endoscopic evaluation of swallowing, see also Sect. 6.2.4) has to be performed. It is a non-invasive dynamic procedure that delivers an immediate evaluation of oropharyngeal swallowing function and directly visualizes the upper aerodigestive tract. However, it has the following limitations: no direct visualization of the bolus on its entire way from the oral cavity to the stomach, no visualization during the swallow, no visualization of the oral and esophageal phases and pharyngo-esophageal segment. Therefore, a videofluoroscopic swallowing study (Ekberg 1992; Logemann 1993, 1998) has to be carried out routinely as a complementary dynamic diagnostic procedure.

2.6.3. Further Examinations

If necessary, further diagnostic methods are used (Table 2.4). Depending on the results of videoendoscopic and videofluoroscopic swallowing studies, esophago-gastroscopy can eventually be performed as a first-line diagnostic method to exclude tumorous lesions or reveal disturbances in the esophageal phase (see also Sect. 6.3). Scintigraphy enables the quantification of bolus transit and aspiration. For an evaluation of neurogenic dysphagia, cranial magnetic resonance imaging (MRI) may detect intracranial lesions responsible for dysphagia. pH-metry represents the gold standard for diagnostics of a suspected gastroesophageal (-pharyngeal) reflux disease. To measure the pressure in the pharynx – especially before surgery of the pharyngo-esophageal segment –, manometry is recommended, which allows measurement of the intrabolus pressures and pharyngeal contraction. Cervical auscultation of the pharyngeal swallow did not gain wide acceptance.

2.7. Conclusion

Swallowing symptoms necessitate an interdisciplinary, thorough diagnostic work-up which reveals aetiologic factors and pathophysiological components. The patient should be asked precise questions relating to symptoms and valuable information should be obtained, allowing the adequate diagnostic and thera-

peutic measures to be taken. Above all, the adequacy of oral nutrition and the presence or absence of aspiration are in the focus of diagnostic and therapeutic interests. Due to an absent cough reflex, aspiration may occur silently. Therefore, diagnostic methods must enable direct visualization of aspiration by videoendoscopy and videofluoroscopy. Aspiration cannot be diagnosed or excluded by patient history or clinical observation alone. However, for an appropriate management, the patient's symptoms should be carefully evaluated.

References

Bastian RW (1991) Videoendoscopic evaluation of patients with dysphagia: an adjunct to the modified barium swallow. Otol HNS 104:339–350

Bigenzahn W, Denk D-M (1999) Oropharyngeale Dysphagien (Oropharyngeal Dysphagia). Ätiologie, Klinik, Diagnostik und Therapie von Schluckstörungen. Thieme, Stuttgart

Brown M, Glassenberg M (1973) Mortality factors in patients with acute stroke. JAMA 224:1493–1495

Denk D-M, Bigenzahn W (1999): Diagnostik oropharyngealer Dysphagien (Diagnostics of oropharyngeal dysphagia). In: Bigenzahn W, Denk D-M (eds) Oropharyngeale Dysphagien. Ätiologie, Klinik, Diagnostik und Therapie von Schluckstörungen. Thieme, Stuttgart, pp 33–65

Denk D-M, Kaider A (1997) Videoendoscopic biofeedback: a simple method to improve the efficacy of swallowing rehabilitation of patients after head and neck surgery. ORL 59:100–105

Ekberg O, Hamdy S, Woisard V, Wuttge-Hannig A, Ortega P (2002) Social and psychological burden of dysphagia: its impact on diagnosis and treatment. Dysphagia 17:139-146

Ekberg O (1992) Radiologic evaluation of swallowing. In: Groher ME (ed) Dysphagia. Diagnosis and management. Butterworth-Heinemann, Stoneham, pp 163-195

Garliner D (1974) Myofunctional therapy in dental practice. Bartel, New York

Groher ME, Bukatman R (1986) The prevalence of swallowing disorders in two teaching hospitals. Dysphagia 1:1–3

Hannig C, Wuttge-Hannig A, Hess U (1995) Analyse und radiologisches Staging des Typs und Schweregrades einer Aspiration (Analysis and radiological staging of type and grade of aspiration). Radiologe 358:741–746

Jones B, Donner MW (eds) (1991). Normal and abnormal swallowing. Springer, Berlin Heidelberg New York

Kuhlemeier KV (1994) Epidemiology and dysphagia. Dysphagia 9:209–217

Langmore S, Schatz K, Olsen N (1988) Fiberoptic examination of swallowing safety: a new procedure. Dysphagia 2: 216–219

Langmore SE, Terpenning MS, Schork A, Chen Y, Murray JT, Lopatin D, Loesche WJ (1998) Predictors of aspiration pneumonia: how important is dysphagia? Dysphagia 13:69–81

Leopold NA, Kagel MA (1996) Prepharyngeal dysphagia in Parkinson's disease. Dysphagia 11:14–22

Logemann JA (1983) Evaluation and treatment of swallowing disorders. Pro-ed, Austin, Texas

Logemann JA (1998) Evaluation and treatment of swallowing disorders. Pro-ed, Austin, Texas

Logemann JA (1993) Manual for the videofluorographic study of swallowing, 2nd edn. Pro-ed, Austin, Texas

Logemann JA (1995) Dysphagia: evaluation and treatment. Folia Phoniatr Logop 47:140–164

Marks B, Schober E, Swoboda H (1998) Diffuse idiopathic skeletal hyperostosis causing obstructive laryngeal edema. Eur Arch Otolaryngol 255:256–8

Miller FR, Eliachar J (1994) Managing the aspirating patient. Am J Otolaryngol 15:1–17

Sasaki H (1991) Management of respiratory diseases in the elderly. Nippon-Kyobu-Shikkan-Gakkai-Zasshi 29:1227–1233

Schröter-Morasch H (1993) Klinische Untersuchung der am Schluckvorgang beteiligten Organe (clinical examination). In: Bartolome G et al. (eds) Diagnostik und Therapie neurologisch bedingter Schluckstörungen. Gustav Fischer, Stuttgart, pp 73–108

Schröter-Morasch H (1996) Schweregradeinteilung der Aspiration bei Patienten mit Schluckstörung (Severity of aspiration in dysphagic patients). In: Gross VM (ed) Aktuelle phoniatrisch-pädaudiologische Aspekte. R. Gross Verlag, Berlin, pp 145–146

3 Imaging Techniques and Some Principles of Interpretation (Including Radiation Physics)

Olle Ekberg

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3.1 Introduction

In the dysphagic patient the radiologic examination of the oral cavity, pharynx and esophagus should be regarded as an extension of the physical and neurologic examinations. The mouth and the pharynx and larynx can be reached only partially during more conventional clinical evaluation. The result of the radiologic examination should thus be put in a broader context together with the clinical history and the result of the clinical and neurologic examinations.

3.2 The Symptom Dysphagia

Dysphagia, i.e., any abnormal sensation during swallowing experienced by the patient, may be caused either by morphologic abnormalities or dysfunction. The clinical history is often spurious and careful work-up must be considered. The swallowing apparatus consists of the oral cavity, pharynx and esophagus and symptoms from any of these three compartments may be present. Morphologic evaluation of these three compartments can be done either by direct inspection (the oral cavity), indirect inspection (the pharynx and larynx), or by endoscopy (the esophagus). However, for a proper functional evaluation radiology is necessary. For the evaluation of transportation through the oral cavity, pharynx and esophagus, the barium swallow is without doubt the most reliable test. It is used extensively and its accuracy has been well shown.

Any clinician interested in patients with dysphagia needs to make sure that his consultant radiologist has the means and interest to perform a proper examination. A carelessly performed examination interpreted as normal may give the clinician a false impression that the examination has been completed and misinterpretation of the barium swallow may lead to unnecessary further work-up and delay correct diagnosis.

3.2.1 Does the Patient Really Have Dysphagia?

When properly performed the radiologic examination should start with a careful penetration of the clinical history. The examination should then be custom-tailored to each patient's specific complaints. The patient who complains of difficulties with certain foods should be examined with such food. The patient who complains particularly of choking during eating must be carefully examined for misdirected swallowing. The patient who complains of heartburn should be examined with respect to gastro-esophageal reflux disease. Patients who complain of pain or obstruction during solid bolus swallow need to be examined with some kind of standardized solid bolus. Therefore, a clinical history is of crucial importance for the radiologist and he needs in his report to state for what symptoms/purposes his study was designed. However,

O. Ekberg, MD
Professor, Department of Diagnostic Radiology, Malmö University Hospital, 205 02 Malmö, Sweden

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it is also important to always examine all three compartments of the swallowing apparatus, namely: the oral cavity, the pharynx and the esophagus. What the clinical history does is to help the radiologist focus on one particular segment and sometimes on a specific food consistency. If the patient has undergone a careful endoscopic examination, before being sent for the radiologic examination, this should not focus on morphology but merely on function. However, most patients with dysphagia do not undergo endoscopy of the esophagus. Therefore, in the majority of the patients with dysphagia, it is important that the radiologist perform both a functional evaluation and a detailed double contrast morphologic examination of the esophagus.

The majority of patients with swallowing symptoms has an abnormality that can be revealed during the radiologic examination whether morphologic or functional. However, many patients are sent for radiologic examinations of the swallowing apparatus without any real swallowing problems. These patients suffer from globus and their spontaneous clinical history always includes complaints or inability to swallow. They feel a sensation of obstruction when swallowing and a constant feeling of a "lump" in the throat. This is the dominant and overwhelming symptom which bothers the patient considerably and this impresses many physicians. However, when a structured clinical history is taken the patient admits that "he can eat and drink normally". This is the hallmark of globus. These patients localize their symptoms to the neck but the symptomatology should alert the radiologist to focus his examination on the lower esophagus in an attempt to reveal signs of gastro-esophageal reflux disease. In fact, these patients, in addition to the inability to swallow "normally" also have heartburn and regurgitation. However, these symptoms are not the leading symptoms and are less alarming than the globus symptom. Therefore, globus should be taken seriously and these patients should be examined properly.

3.2.2 Findings Compared to Symptoms

The careful clinical history also serves another purpose, namely to make an assessment of the clinical relevance of any radiologic finding. A comparison of the patients symptoms and the radiologic finding should always be included in the radiologist's report. The radiologic examination may reveal a host of abnormalities, most of which are irrelevant in that particular clinical setting. In our experience the radiologist, experienced in dysphagia evaluation, is best suited to make this comparison between symptoms and findings. The radiologist knows how the examination was performed and may also add to the clinical history after the examination is done. It may be true that many patients with dysphagia do not undergo a proper functional evaluation of the swallowing apparatus. However, the result of many barium studies is at the same time misinterpreted and not put into a proper clinical context.

3.2.3 Dysphagia During the Radiologic Examination

As well as the clinical history concerning symptoms and signs is important, it is as important to register symptoms during the radiologic examination. If misdirected swallowing occurs, the radiologist needs to observe if cough or other signs of the patient's subjective experience of that misdirected swallowing event occurs. By and large, patients who do not cough during even minor events of misdirected swallowing have a more advanced disease than those who cough and an increased risk of airway disease (Fig 3.1, 3.2). If a tablet is given to evaluate solid bolus dysphagia and if the patient does experience symptoms similar to the ones he experienced during normal eating and drinking the tablet test can be considered diagnostic (van Westen and Ekberg 1993). However, even if the tablet gets stuck in the esophagus (excluding patients with strictures) for many minutes and the patient does not experience this, the tablet test has not revealed the cause of that particular patient's dysphagia. However, one needs to remember that patients usually do not comment spontaneously about symptoms during the test. This is often due to the fact that symptoms provoked during the radiologic examination are much milder than during normal eating and drinking. This circumstance should alert the radiologist to carefully but casually ask the patient about swallowing symptoms during this test.

3.3.1 The Radiologic Examination (Barium or Iodine Swallowing)

In the team of professionals taking care of the neurologically impaired patients with swallowing dif-

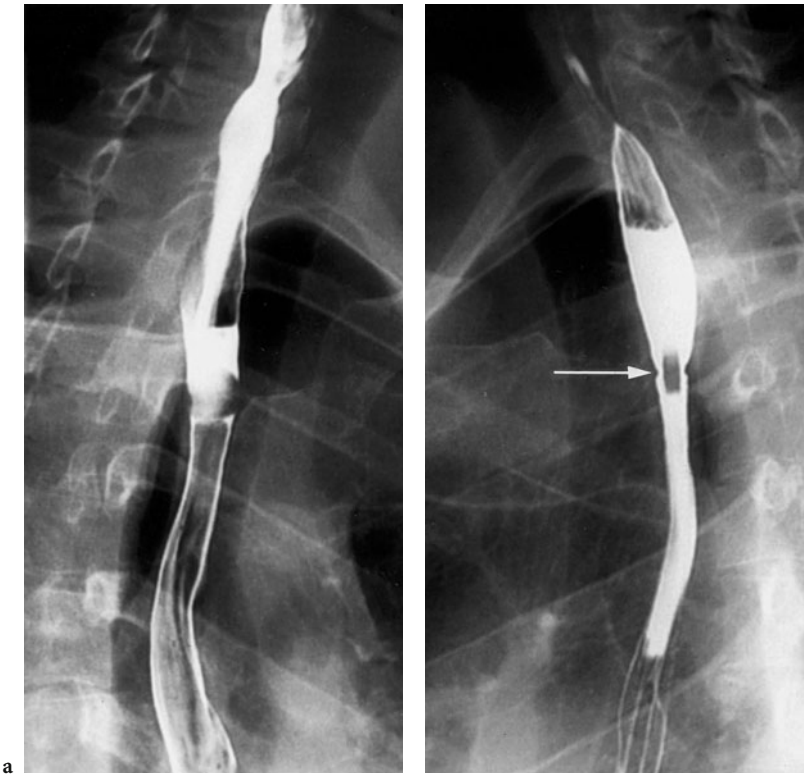


Fig. 3.1a,b. A 46-year-old woman with solid bolus dysphagia. The initial barium examination was assessed as normal. A 13-mm in diameter antacid tablet used for stressing the esophagus got stuck in the proximal esophagus. A short asymmetric narrowing (assessed to be congenital in origin) was revealed by the tablet. Balloon dilatation made the patient become asymptomatic

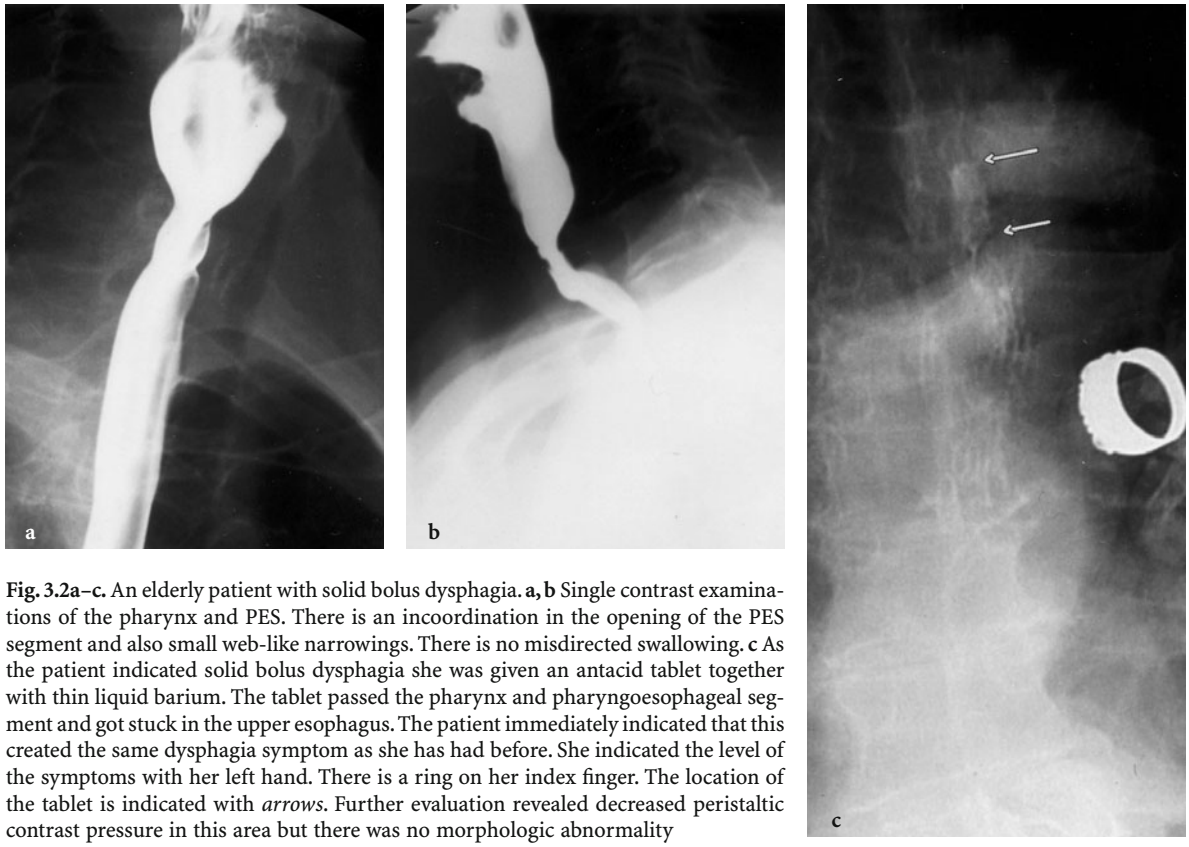


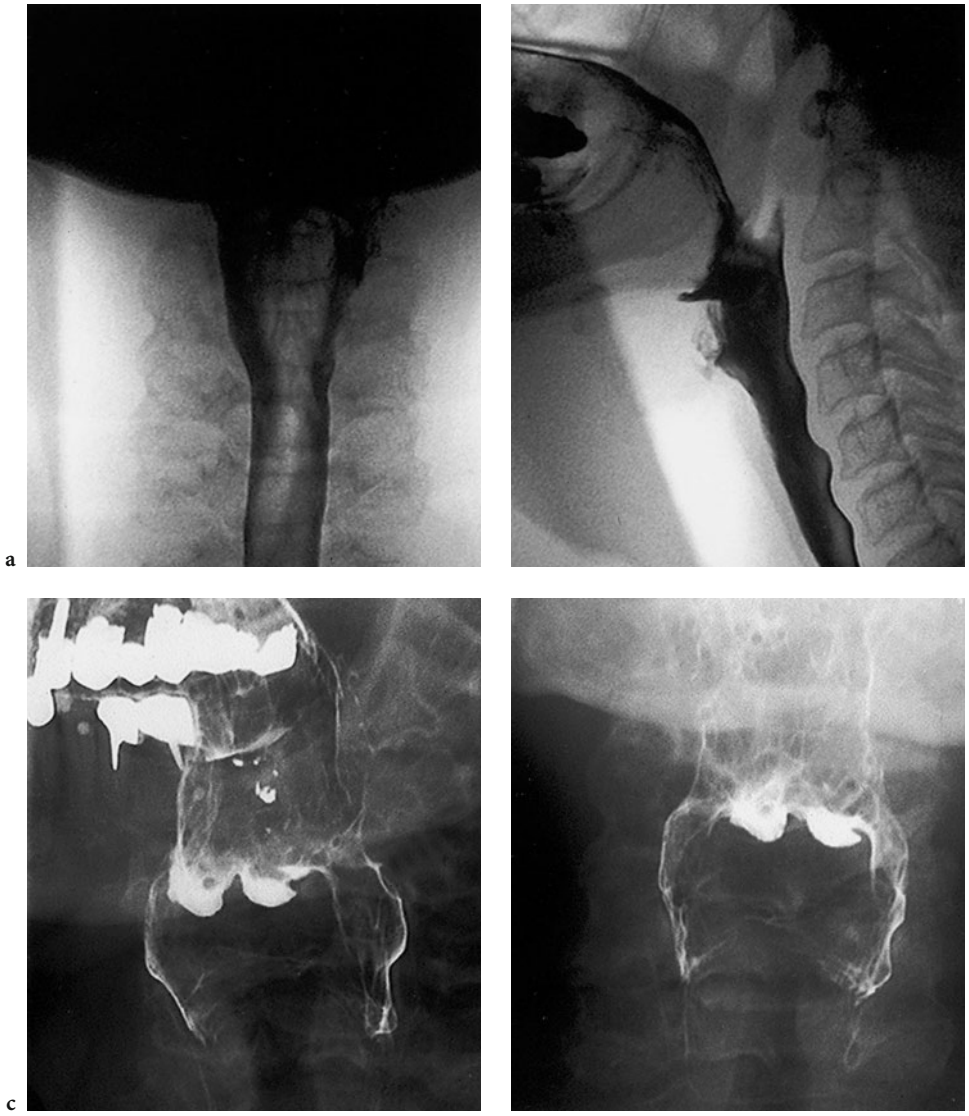
Fig. 3.2a–c. An elderly patient with solid bolus dysphagia. **a, b** Single contrast examinations of the pharynx and PES. There is an incoordination in the opening of the PES segment and also small web-like narrowings. There is no misdirected swallowing. **c** As the patient indicated solid bolus dysphagia she was given an antacid tablet together with thin liquid barium. The tablet passed the pharynx and pharyngoesophageal segment and got stuck in the upper esophagus. The patient immediately indicated that this created the same dysphagia symptom as she has had before. She indicated the level of the symptoms with her left hand. There is a ring on her index finger. The location of the tablet is indicated with *arrows*. Further evaluation revealed decreased peristaltic contrast pressure in this area but there was no morphologic abnormality

ficulties the radiologist plays an important role. He must choose the correct imaging technique which will most expediently and accurately answer the clinical questions. This is not always barium swallow. For instance in patients with the combination/constellation of neck pain and dysphagia MRI can reveal soft tissue disease, such as retropharyngitis (Ekberg and Sjöberg 1995). The radiologist in the swallowing team must be aware of the limitations of radiology for assessment of the course and treatment of the disease process.

Radiology therefore offers a unique possibility to screen patients with swallowing symptoms. The barium swallow allows evaluation of both morphology and function, which is unique to this technique.

3.3.1.1 How to Perform the Barium Swallow Examination

The radiologic examination should be performed as a biphasic examination, i.e., both function (single contrast) and morphology (double contrast) should be evaluated (Fig. 3.3). All patients with dysphagia can be accurately examined radiologically! Even in the severely ill stroke patient the relevant clinical questions can be answered. In the very impaired patient it is often enough to demonstrate whether or not the patient can elicit the pharyngeal stage of swallowing and maybe also the degree of aspiration if such occurs. Such an examination is usually easy to perform. Cumbersome and time consuming are



e

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g

h

i

Fig. 3.3a–i. Biphasic examination of the pharynx and esophagus. **a, b** Frontal and lateral projections of the pharynx. These images are digitally stored from the fluoroscopic images. **c** Double contrast examination of the pharynx in slight left anterior oblique LAO projection. **d** Frontal projection of the pharynx, double contrast. **e** Right anterior oblique projection of the pharynx examined with double contrast technique. **f** Lateral double contrast examination of the pharynx. **g–i** Double contrast examinations of the esophagus in different projections

examinations in ambulatory, alert, otherwise healthy young patients who complain of vague or uncharacteristic symptoms during swallowing.

Crucial components of the radiologic equipment for evaluation of patients with dysphagia is a video recorder. Such a video recorder can be of any low-cost consumer model. For $500 the radiologic examination will be converted from a meaningless spot film evaluation of static images to a full-blown dynamic imaging modality. More costly equipment will increase the band width and thereby the resolution of the images. However, such an investment will only add marginally to the diagnostic capability. Much more important is to install the video recorder so as to record only during fluoroscopy. It should automatically be controlled by the fluoroscopic pedal. The video recorder must also have the possibility to review the tape on a frame-by-frame mode and also to review the tape forward and backward. This should be controlled by a jog-wheel.

The image should be centred at the important area whether oral cavity, pharynx or esophagus. Moreover, always use proper cone down, and most importantly, use collimation. If wedge frames are available: always use them. Always use a high voltage (110 kV), as this will shorten the exposure time, thereby minimising movement artefacts.

Do not move the fluoroscopic intensifier during exposure. Also keep it steady during fluoroscopy. Moving around the image-intensifier may save contrast medium but deteriorates image sharpness, thereby losing important morphologic information (Fig. 3.4).

Modern digital radiologic equipment offers the possibility to store the fluoroscopic image in digital form (Figs. 3.5–3.7). This brings down the radiation dose to the patient (and the radiologist!).

It is important to perform fluoroscopy and obtain images in standardised projections. Fluoroscopy is used for assessment of function but also for positioning. Especially in the pharynx the importance of meticulous positioning and projection is important (Fig. 3.8–3.10).

All patients should be examined in an erect sitting or standing position if possible. We prefer to use high density barium (240 w/v) for the double contrast evaluation of the oral cavity, pharynx and esophagus. The high density of the barium also makes it relatively easy to register penetration of barium into the laryngeal vestibule (Fig. 3.11). However, such high density barium, probably due to its high viscosity, is less likely to reach into the vestibule in patients with defective closure. Therefore, sometimes penetration is revealed only using

Fig. 3.4. The distal part of the esophagus filled with barium and air. The image intensifier was moved during the exposure and this created unsharpness

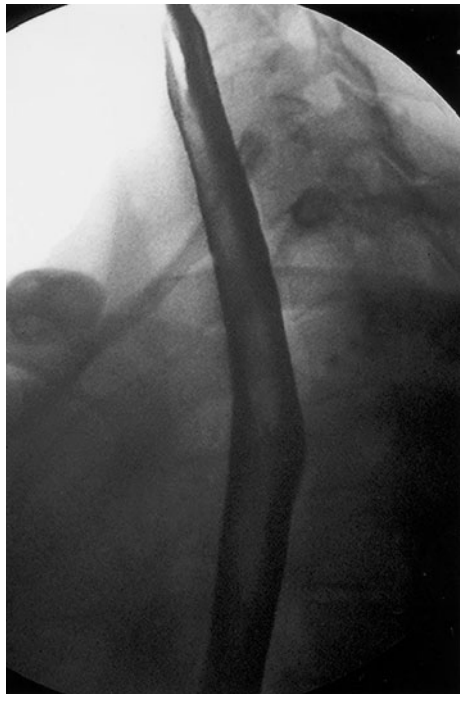


Fig. 3.5. Digitally stored image of the esophagus. This gives an image with inferior spatial resolution compared to an exposed image, but the radiation dose is much lower

Fig. 3.6a,b. The difference in image quality between conventional (digital) exposure technique and the digital storage of a frozen fluoroscopic image is illustrated in this patient. **a** Exposure technique; **b** digitally stored fluoroscopic image. The spatial resolution is higher with an exposure technique. This is particularly important when assessing morphologic abnormalities. However, the detection of minor amounts of misdirected swallowing into the laryngeal vestibule can also be difficult using fluoroscopic technique with digital storage only. However, the reduction in radiation dose is substantial

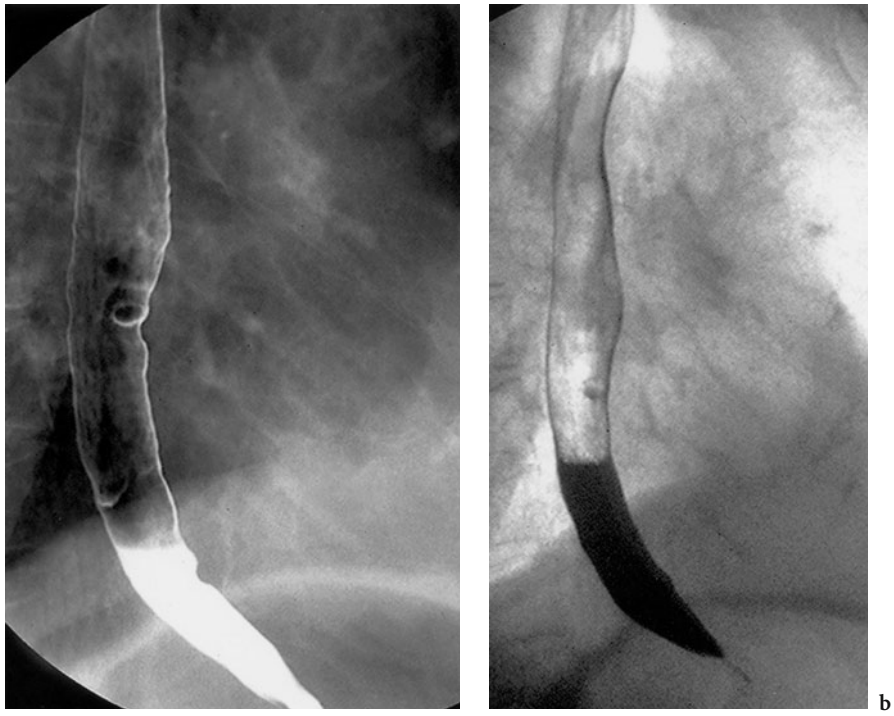


Fig. 3.7a,b. Comparison between different techniques. The exposure in (**a**), and storage image of the frozen fluoroscopic image in (**b**)

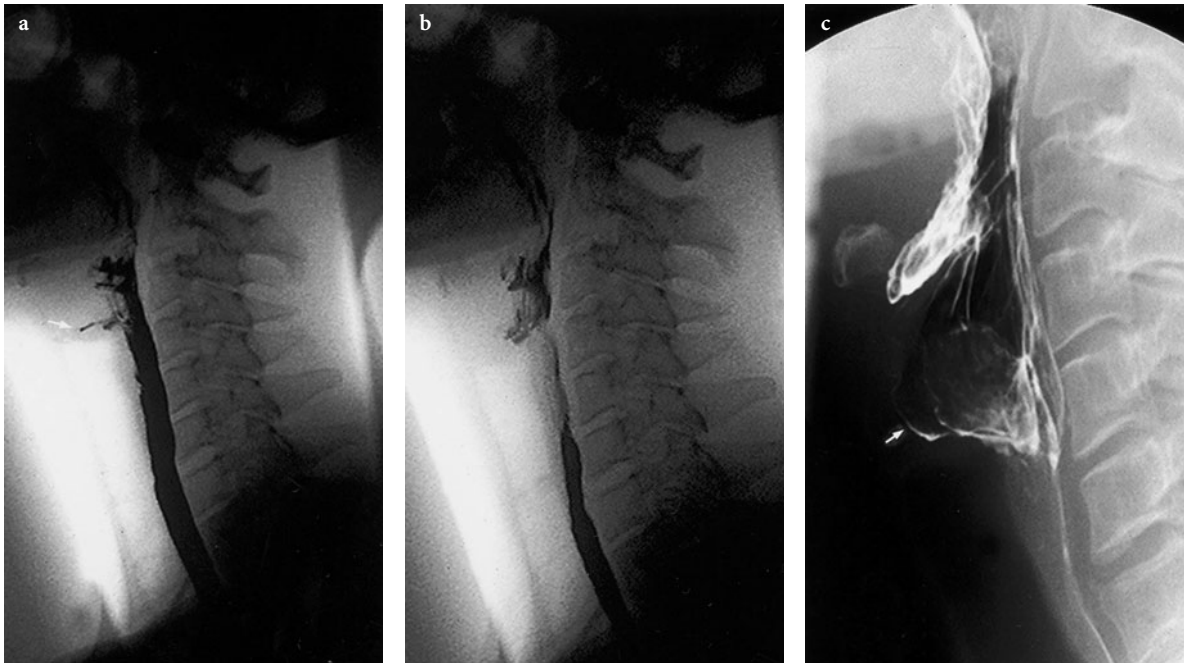


Fig. 3.8a–c. The importance of correct projections/positioning. This patient was assessed to have contrast medium reaching into the laryngeal vestibule (*arrow*) in (**a**). Contrast medium in one of the piriform sinuses in this slightly oblique projection mimics contrast medium in the laryngeal vestibule. However, this image was obtained in a slightly oblique projection. This was revealed during a more precise positioning of the patient in (**b**). **c** The same mistake can easily be made during double contrast examination when contrast medium in one of the piriform sinuses may mimic contrast medium in the laryngeal vestibule (*arrow*)

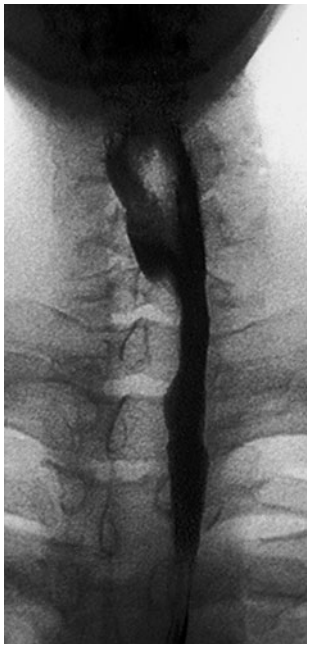


Fig. 3.9. Frontal projection of the cervical esophagus during barium swallow. There is a fusiform indentation from the right into the esophagus. This is the characteristic appearance of the effect of the kyphosis of a cervical spine. The esophagus is stretched over the kyphosis and deviates to the left. This gives a false impression of a "pseudotumour"

a low density barium (40% w/v). However, with the proper exposure technique even minute amounts of such contrast medium should be possible to reveal.

3.3.1.2 How to Perform the Iodine Swallow Examination

Only in patients who are strictly NPO (non-peroral feeding) it is indicated to replace barium with iodine contrast medium. The basic advantage with barium contrast medium is that it is radiopaque. It is also not harmful for the mucosa in the airways. It is readily transported cranially in the airways by the cilia. However, in patients with emphysema and other chronic obstructive lung diseases the transportation might be delayed and even absent from the most distal parts, i.e. the alveoli and emphysematous bullae. In such patients it might also be indicated to use iodine contrast medium. Iodine contrast medium generally used to be a hyperosmotic fluid with a very strong taste and smell. Nowadays the iodine is usually presented as non-ionic, isoosmolar or slightly higher compared to plasma. Usually it has a sweet taste and no smell. The radiodensity of such a contrast medium (e.g. 350 mg I/ml) is appropriate for evaluation of morphology and function. In a patient where iodine contrast medium has been chosen

a

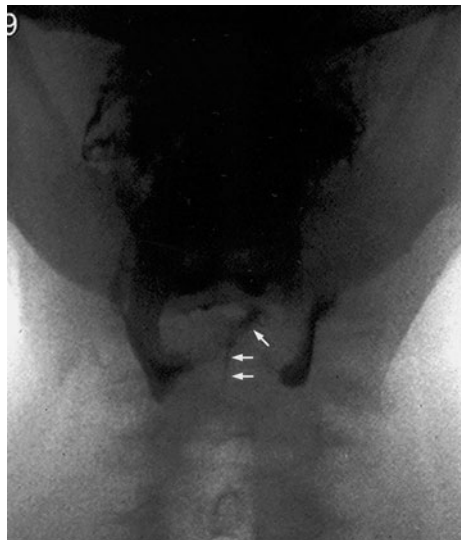
b

c

d

Fig. 3.10a–d. a A single contrast examination of the pharynx. The folded appearance of the epiglottis which creates a hole in the contrast medium. This image also shows the medialisation of the lateral pharyngeal wall as an indicator of normal function of the constrictors. **b** This is much more difficult to assess in lateral projection. **c** The characteristic, irregular in-bulging of the arytenoid region and the down-folded epiglottis is seen creating an irregular impression in the anterior wall of the midpharynx (*arrow*). **d** For proper evaluation of the pharyngoesophageal segment it is often necessary to obtain images in the oblique projection

a



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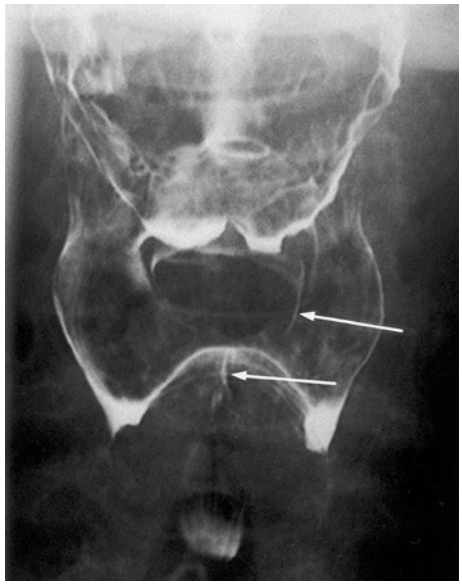

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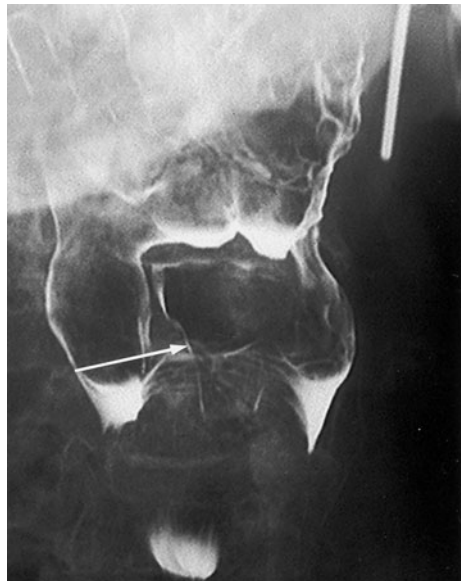

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Fig. 3.11a–e. It is important to be able to identify misdirected swallowing also in a frontal projection. During the full column examination, also called single contrast technique, the laryngeal vestibule is obscured in the frontal projection. **a** The oblique epiglottis (*arrow*) can be seen. This is per se not a functional abnormality. **b** Retention of barium in the pharynx showing that there is also contrast medium outlining the characteristic shape of the half-opened laryngeal vestibule (*arrows*). The shape resembles a tapered glass. **c** Contrast medium on the inside of the laryngeal vestibule up to the aryepiglottic folds (*arrows*). There is also contrast medium between the opposed arytenoids (*lower arrow*). The same is seen in LAO (**d**), and RAO (**e**) projections. Barium seen in the laryngeal vestibule, as in this patient, indicates misdirected swallowing. This is always abnormal but may or may not be symptomatic

as the contrast medium, the clinical situation is usually a severely disabled patient with a very limited number of relevant clinical questions. One such question can be whether or not the patient can elicit a pharyngeal swallow. Therefore, the projections and number of swallows is much more individualised. It is usually sufficient to observe one or only a few swallows and usually in lateral projection. Although double contrast films may also be obtained with iodine contrast media, this is usually not indicated. However, even in a very limited examination it is important to ascertain that there is no obstruction for the bolus. Therefore it is important to at least fluoroscopically examine the pharynx, PES and esophagus in search for tumours or strictures. In a supine position, which is usually the only position that the patient can take, ample evaluation of esophageal motor function is also possible.

Another situation where iodine contrast medium is regularly used is in patients where there is a question of a leak and also in a postoperative situation where patency of anastomosis is evaluated (see Sect. 4.4).

3.3.2 The Oral Cavity and Pharynx

As in most clinical circumstances the question of misdirected swallowing is the most crucial one and this should be carefully looked into. The patient should be in a lateral projection, preferably in an erect position, although the patient may also be recumbent. The field of imaging should include the oral cavity, soft palate, pharynx and the pharyngo-esophageal segment. In that position, the patient should be instructed to take a mouthful of liquid barium into the mouth. The size of the ingestion should be according to his own discretion. The radiologist should take time to explain this part of the examination. The volume ingested should then be evaluated and compared to the patient's swallowing capability. The ingested material should be observed in the oral cavity for at least 5–10 s. Any anterior or posterior leak should be observed during that period. It should also be possible to assess the volume of ingestion as many patients with oral apraxia or defective sensitivity are ingesting too big a bolus to be handled safely during swallowing. The fluoroscopy should be kept on during the ingestion and until the bolus tail has passed into the cervical esophagus. Collimation and exposure techniques should be meticulously controlled and if possible wedged collimation should be used. This decreases flaring due to the air between the chin and the neck. A minimum of three swallows should be registered in this latter projection. However, if misdirected swallowing is a major concern and such has not been revealed during these three swallows it is important to observe additional swallows. We may observe at least 15 swallows in such patients (and even more if necessary). During such swallows provocation may be added. Patients may be able to compensate for defective closure of the laryngeal vestibule and/or misdirected swallowing. By altering the position of the head and neck such a compensation may be decompensated and thereby the cause of the patient's complaints explained (Ekberg 1986). The easiest way to decompensate is to ask the patient to swallow with his/her neck extended. Patients with normal function should be able to close their laryngeal vestibule and misdirected swallowing should not occur during swallowing even if the neck is extended. Patients who have partial malfunction may decompensate during this manoeuvres.

We have found that it is much easier to assess the video recording if a soundtrack of the same is also recorded during the examination. Therefore we have installed a microphone conveniently mounted on the X-ray shield. During the video recording all pertinent information such as bolus viscosity, bolus volume, patient positioning and perhaps most importantly any symptoms reported by the patient during the examination is recorded and can easily be assessed during the review if a loudspeaker is included in the reading room equipment.

3.3.3 The Esophagus

For a detailed description of how to examine esophageal function, please see Sect. 5.1 Below is a short introduction. It is of the utmost importance to always include all these compartments (the oral cavity, pharynx and esophagus) in the radiologic examination. However, in each patient focus is usually on only one or two compartments.

We prefer to perform the double contrast examination of the esophagus after the functional evaluation of the pharynx. The esophageal examination starts with effervescent tablets and the patient is asked to rapidly drink the high density barium (240% w/v). Double contrast radiograms are taken in left and right oblique projections. Patients with painful swallow should be evaluated for esophagitis such as *Candida*. The radiologist should always be careful in evaluating for neoplastic lesions such as superficial carcinomas.

Evaluation of esophageal function often needs to be done in a recumbent position. However, if esophageal dysfunction is already revealed in an erect position it is usually not warranted to place the patient recumbent as dysfunction in the erect position is not known to become normal in the recumbent position.

3.4 Solid Bolus Examination

All patients with a history of obstruction during swallowing should be examined with a solid bolus, especially if solid bolus dysphagia is present (see Figs. 3.1, 3.2). The solid bolus used for the radiologic examination must be standardised. We prefer to use a radiolucent antacid tablet with a known diameter of 13 mm (van Westen and Ekberg 1993) This is swallowed together with thin liquid barium (40% w/v). Others have advocated the use of a barium tablet, a bread sphere, or a marshmallow (Curtis et al. 1987; Kelly 1961; Somers et al. 1986). Contraindications for tablet swallowing are known or suspected pharyngeal dysfunction with misdirected swallow. If this is suspected, the patient should first undergo evaluation of the pharyngeal stage and if misdirected swallowing is present with barium reaching below the vocal folds the solid tablet should not be given. This is because of the risk of choking. However, the best way to triage patients for the tablet test is to show them the tablet and the thin liquid barium and ask them if they can swallow the tablet. In our experience this easily excludes patients who should not undergo the tablet swallow. In fact that particular group of patients who do not think they can swallow the tablet are the ones who should be examined due to suspected stricture or other obstructions.

Tablet arrest may or may not be symptomatic. When the tablet gets stuck above a stricture, symptoms are usually absent. This is in contrast to when the tablet arrest is due to dysmotility. In the latter cases the patient is often symptomatic. However, the symptom is often much milder than the symptoms that arise during a meal.

Solid bolus arrest in the esophagus is either due to dysmotility or to a narrowing. Dysmotility is either due to spasm or hyperperistalsis, two entities that are hard to separate fluoroscopically. As assessed by manometry most of these patients have hypomotility and only rarely spasm.

3.5 Aspects of Radiation Physics/Safety

Radiologic examination relies on the ability of photons of a certain energy (the X-rays), to penetrate opaque materials such as the human body. Since their discovery by Wilhelm Conrad Röntgen in 1895, these have made a major contribution to medicine as well as other industrial applications. However, such radiation means that energy is transported through the body. Only 1%–20% of the given energy from the X-ray tube reaches the detector, i.e. the image intensifying screen or the film screen combination. The rest of the energy is absorbed in the patient. It is this absorbed energy, which is sometimes called radiation dose, that causes the main hazardous aspect of radiation. Such ionised radiation, when absorbed in the body, might interfere with water molecules and cause free radicals. The ionising radiation may also interfere with proteins and other important substances within the cell. This reaction may cause cell death or induce serious late side effects. It is therefore necessary that all radiation, including the one used for swallowing studies, is used with care.

One important aspect to remember is that it is only when the X-ray tube is irradiating, namely when the current is closed, that there is radiation from the tube and in the room. At the same moment as the current is broken, the radiation primarily from the X-ray tube (secondarily from the patient) stops and there is no hazard within the room or to the patient, nor to the personnel in the room. Radiation always and only traverses linearly, i.e. it does not bend around corners.

The X-ray equipment must undergo regularly scheduled quality controls according to each country's regulations. It is also important that personnel responsible for the equipment ascertain that all functions are checked for proper function on at least a weekly basis.

The most important consideration is that an X-ray examination should never be performed unless it is necessary. It should be contemplated beforehand what the referring clinician is expecting to find and what therapeutic or prognostic consequences that may have. Once this is clear the risk for radiation damage is always far smaller than the benefit that can be achieved from the X-ray examination. This means that the X-ray examination needs to be necessary in the clinical setting and then that the performance of the study must be optimised. The risk for damage is far greater in children and young adults. In individuals over 40, the risk is basically negligible. There is

a positive correlation between patient dose and personnel dose. It is important to cone down on the pharynx and/or esophagus and thereby avoid radiation of extensive volumes. The distance between the patient and the image intensifier/screen should be as small as possible.

Try to stay as far away as possible from the patient. However, be close enough for assistance or manipulations. Wear lead aprons! Sometimes an apron over the thyroid or lead eyeglasses have been advocated. Most importantly, if you do not need to stand close to the patient: step away. If you double the distance between the patient and yourself, the radiation dose goes down to one quarter. Remember that it is not the X-ray tube that gives radiation to the personnel in the room, but it is the patient who radiates secondary radiation. It is also important to carefully perform the study, meaning that it is better to do one proper radiologic examination than to have to re-do studies or even exposures due to bad technique. It is also important to realise the fluoroscopy time that contributes to the majority of the radiation dose. A 1-minute fluoroscopy is roughly equivalent to one exposure. This is however relative and depends on the situation. In a study by Chan et al. in Hong Kong they found that if a videoradiographic swallowing study had a duration of 18±8 min (and this is very long from my own perspective) the scattered radiation dose (measured 30, 60 and 100 cm from the patients neck) to the examiner varied tenfold, i.e. 34–3 μSv (Chan et al. 2002). This should then theoretically permit an examiner to perform 2583 examinations/year given the annual effective dose limit of 20 mSv. It should be noted that since lead aprons are always worn, the additional protection they provide further reduces the scattered radiation dose to negligible levels.

If you have access to digital radiology, you may also have access to pulsed fluoroscopy. If possible, use as few pulses per second (usually two) as possible. However, this might sometimes be unsuitable for evaluation of function. It is better to use continuous fluoroscopy than to have to re-do the swallowing studies. Use as short as possible fluoroscopy time. Be sure that the patient does not move around and if necessary, have the patient to hold his breath during exposures. Avoid flaring by using wedge frames. Modern equipment also has the possibility to save the fluoroscopy image digitally. This may in fact lower the dose considerably in comparison to conventional exposures. These images may be enough for documentation of for instance misdirected swallowing. However, as stated above, it is better to make one proper exposure and one properly performed examination than to have to re-do the study due to poor image quality. If studies are performed by speech-and-language pathologists it is very important that they are familiar with radiation physics.

The effective dose of a radiologic dysphagia evaluation as described above is about 1 mSv. This could be compared to a chest X-ray (AP and lateral): 0.1 mSv; abdominal survey film (three images): 5 mSv; barium colon examination: 5 mSv; coronary angiography with interventions as well as interventions in other parts of the body may easily exceed 100 mSv.

3.6 Other Techniques

Ultrasound can be used for evaluation of the oral stage and hyoid bone movement (Hamlet et al. 1988; Brown and Sonies 1997). This technique is attractive because it does not rely on ionising radiation. It can therefore be used unrestricted also in younger patients. It also enables visualisation of the tongue surface. It could be the method of choice for monitoring oral function. However, the fact that the bolus is not visualised is a major drawback. It also requires considerable operator skills.

During recent years there has been a growing interest in fibre-endoscopic evaluation of swallowing (FEES). During FEES the endoscope is inserted through one of the nostrils and the pharynx and larynx are observed before and after swallow (Langmore et al. 1988). Any posterior leak before swallowing over the tongue base can be observed, as well as movement of the pharyngeal wall. Also, laryngeal structures during breathing are observed. Sensitivity may be tested by touching the walls of the pharynx with the tip of the fibre endoscope. When the pharynx is elevated during swallowing and the bolus is brought into the pharynx the endoscopist's view is obstructed. However, retention in the pharynx after swallowing can be observed and also whether or not bolus has reached into the larynx. As a bolus one usually uses water intensely coloured in order to better visualise the same.

The ROSS (repetitive oral suction and swallowing) test was introduced in the mid-1990s. Using a multimodality and simultaneous technique (ultrasound, pressure, scale, temperature) it was possible to measure suction pressure, bolus volume, respiration and feeding-respiratory co-ordination, and pharyngeal transit time (Nilsson et al. 1995).

A "swallowing safety index" could also be calculated (Nilsson et al. 1997). This technique has so far not gained widespread use.

The most promising "new technique" is MRI. With fast gradients and short acquisition times an "almost real-time" fluoroscopic technique is possible. This allows analysis of bolus transport but also, and more importantly, movements in anatomical structures, i.e. the tongue and pharyngeal constrictors (Buettner et al. 2001).

3.6.1 Videomanometry

Dynamic cine- and videoradiography of pharyngeal barium swallow provides morphological as well as qualitative functional information of the swallowing sequence. However, dynamic barium swallow relies mainly on functional qualitative judgement, failing to quantify the results. This is for example the case when trying to assess pharyngeal paresis in terms of degree of barium retention in the pharynx.

In this context, intraluminal pharyngeal manometry is capable of providing a more quantitative analysis of pharyngeal muscle function in terms of intraluminal pressure registration (McConnel 1988, McConnel et al. 1988a, 1988b; Olsson and Ekberg 1995; Olsson et al. 1994, 1995, 1996). Concurrent barium swallow and pharyngeal manometry combine qualitative assessment of bolus transport with quantitative registration of the contractions of the pharyngeal wall. Our experiences with this simultaneous technique have revealed that there is a substantial longitudinal asymmetry in pharyngeal pressure response in normal individuals. There is also a wide variety in the timing of PES opening/relaxation, where the pressure registration is extremely dependent on sensor positioning. Furthermore, the radiologic finding of a CP bar is often only an indicator of a more widespread dysfunction around the PE segment easily evaluated with the simultaneous technique. In many patients with a posterior CP bar the major abnormality is weak constrictors with outpouching of the lumen above and below.

The simultaneous technique provides new diagnostic information in dysphagic patients and we suggest the addition of pharyngeal solid state manometry, preferably with simultaneous videoradiography, in cases where routine radiologic work-up does not reveal any pathology.

References

Brown BP, Sonies BC (1997) Diagnostic methods to evaluate swallowing other than barium contrast. In: Perlman AL, Schulze-Delrieu KS (eds) Deglutition and its disorders. Anatomy, physiology, clinical diagnosis, and management. Singular Publ. Group, San Diego, pp 227–253

Buettner A, Beer A, Hannig C, Settles M (2001) Observation of the swallowing process by application of videofluoroscopy and real-time magnetic resonance imaging--consequences for retronasal aroma stimulation. Chem Senses 26:1211–1219

Chan CB, Chan LK, Lam HS (2002) Scattered radiation level during videofluoroscopy for swallowing study. Clin Radiol 57:614–616

Curtis DJ, Cruess DF, Willgress ER (1987) Abnormal solid bolus swallowing in the erect position. Dysphagia 2:46–49

Ekberg O, Sjöberg S (1995) Neck pain and dysphagia. MRI of retropharyngitis. J Comp Assist Tomogr 19:555–558

Ekberg O (1986) Posture of the head and pharyngeal swallow. Acta Radiol Diagn 27:691–696

Hamlet SL, Stone M, Shawker TH (1988) Posterior tongue grooving in deglutition and speech: preliminary observations. Dysphagia 3:65–68

Kelly JE Jr (1961) The marshmallow as an aid to radiologic examination of the esophagus. N Engl J Med 265:1307–1307

Langmore SE, Schatz K, Olsen N (1988) Fiberendoscopic examination of swallowing safety: a new procedure. Dysphagia 2:216–219

McConnel FMS (1988) Analysis of pressure generation and bolus transit during pharyngeal swallowing. Laryngoscope 98:71–78

McConnel FMS, Cerenko D, Hersh T, Weil LJ (1988a) Evaluation of pharyngeal dysphagia with manofluorography. Dysphagia 2:187–195

McConnel FMS, Cerenko D, Jackson RT, Hersh T (1988b) Clinical application of the manofluorogram. Laryngoscope 98: 705–711

Nilsson H, Ekberg O, Olsson R, Hindfelt B (1995) Oral function test for monitoring suction and swallowing in the neurologic patient. Dysphagia 10:93–100

Nilsson H, Ekberg O, Bülow M, Hindfelt B (1997) Assessment of respiration during video fluoroscopy of dysphagic patients. Acad Radiol 4:503–507

Olsson R, Castell JA, Castell DO, Ekberg O (1995) Solid-state computerized manometry improves diagnostic yield in pharyngeal dysphagia: simultaneous videoradiography and manometry in dysphagia patients with normal barium swallows. Abdom Imag 20:230–235

Olsson R, Ekberg O (1995) Videomanometry of the pharynx in dysphagic patients with a posterior cricopharyngeal indentation. Acad Radiol 2:597–601

Olsson R, Kjellin O, Ekberg O (1996) Videomanometric aspects of pharyngeal constrictor acitivity. Dysphagia 11:83–86

Olsson R, Nilsson H, Ekberg O (1994) Pharyngeal solid-state manometry catheter movement during swallowing in dysphagic and nondysphagic participants. Acad Radiol 1:339–344

Somers S, Stevenson GW, Thompson G (1986) Comparison of endoscopy and barium swallow with marshmallow in dysphagia. J Can Assoc Radiol 37:72–75

van Westen D, Ekberg O (1993) Solid bolus swallowing in the radiologic evaluation of dysphagia. Acta Radiol 34:332–375

4 Pharynx

Olle Ekberg, Stephen Rubesin, Rolf Olsson, Christian Hannig, Anita Wuttge-Hannig, Margareta Bülow, Bonnie Martin-Harris

4.1 Oral and Pharyngeal Function and Dysfunction

Olle Ekberg

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4.1.1 Introduction

Normal pharyngeal swallow is precisely scheduled and symmetric. It is finely tuned and coordinated in a precise and exact manner to establish a safe swallow (Miller 1986; Dodds 1989). The swallowing process is regulated by a command centre in the brainstem, a central program generator which receives input from the cerebral cortex and peripheral muscle and directs the sequence of swallowing. This process is both voluntary and involuntary and incorporates motor activity from the oral cavity, pharynx and esophagus. It involves both motor and sensory activity. There is an evolving amount of knowledge concerning normal and abnormal swallowing (Jones and Donner 1991; Ekberg and Wahlgren 1985; Hannig and Hannig 1987; Brühlmann 1985; Pokieser et al. 1995; Dodds 1989).

O. Ekberg, MD
Professor, Department of Diagnostic Radiology, Malmö University Hospital, 205 02 Malmö, Sweden

4.1.2 The Normal Swallow

4.1.2.1 The Oral Stage

The oral, pharyngeal, pharyngoesophageal segment (PES) and esophageal stages of swallowing are readily appreciated radiographically (Fig. 4.1.1). The oral stage of swallowing is bolus specific, i.e. the patient handles different boluses differently, i.e. a strawberry is handled differently from a cup of tea. Therefore, the oral stage is notoriously more difficult to evaluate radiologically than the rest of the swallowing apparatus. However, the oral stage should be included in the radiologic evaluation. The recording should start with the ingestion. During oral processing there is superior and inferior and some anterior-posterior movement of the hyoid bone. However, liquid barium should not be processed or modified in the oral cavity. Therefore, oral preparation of swallow is tested with a solid or semi-solid bolus. When the ingested material is ready to be swallowed the same is brought on to the back of the tongue, which obtains the shape of a groove (Hamlet et al. 1988). This is the swallow preparatory position.

No part of the bolus is allowed to leak anteriorly from the mouth through the lips. Even more important radiologically is to observe if posterior leak occurs. The patient should be able to control the sealing of the tongue base to the soft palate and posterior pharyngeal wall.

4.1.2.2 The Pharyngeal Stage

The pharyngeal swallow is initiated voluntarily and the material to be swallowed is usually called bolus from this point. Initiation of the pharyngeal swallow coincides with the beginning of the anterior movement of the hyoid bone from an elevated position. Pharyngeal constriction is probably cued by bolus interfering with sensory innervation at the faucial

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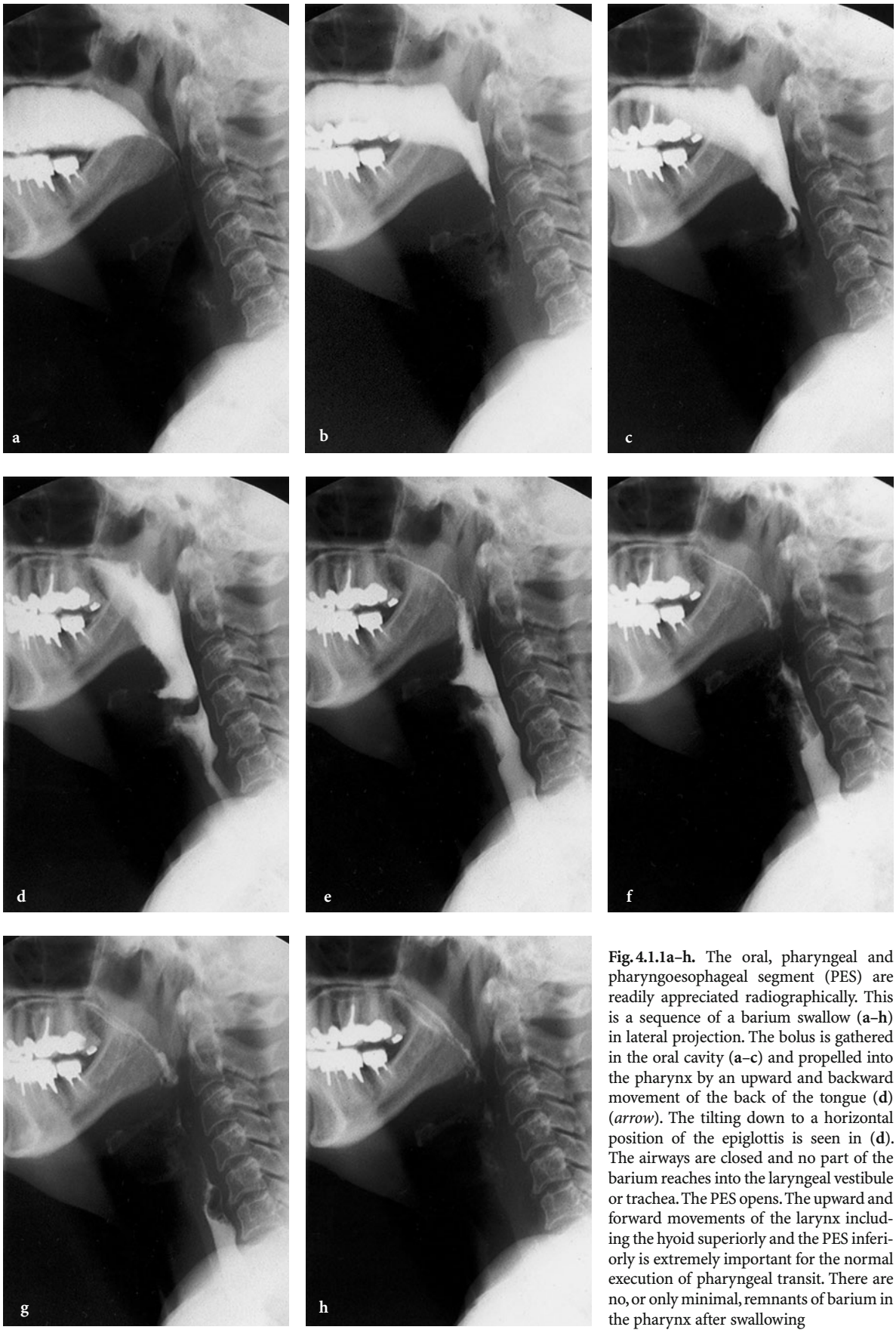


Fig. 4.1.1a–h. The oral, pharyngeal and pharyngoesophageal segment (PES) are readily appreciated radiographically. This is a sequence of a barium swallow (**a–h**) in lateral projection. The bolus is gathered in the oral cavity (**a–c**) and propelled into the pharynx by an upward and backward movement of the back of the tongue (**d**) (*arrow*). The tilting down to a horizontal position of the epiglottis is seen in (**d**). The airways are closed and no part of the barium reaches into the laryngeal vestibule or trachea. The PES opens. The upward and forward movements of the larynx including the hyoid superiorly and the PES inferiorly is extremely important for the normal execution of pharyngeal transit. There are no, or only minimal, remnants of barium in the pharynx after swallowing

isthmus. Radiologically it is convenient to use the beginning of the anterior hyoid movement as a starting point of pharyngeal swallow. The tongue then propels the bolus posteriorly into the pharynx and further down into the PES and cervical esophagus. If the pharyngeal constrictor wall has normal compliance only minor dilatation of the pharynx occurs (Fig. 4.1.2).

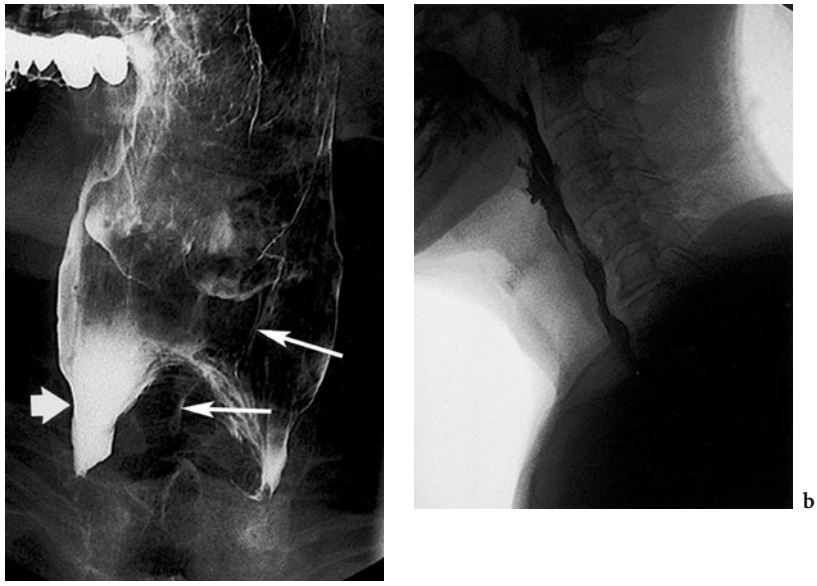
The palatopharyngeal isthmus is closed by elevation of the muscular palate and constrictor convergence which is most medialward of the lateral walls. Normally, no regurgitation of barium into the nasopharynx occurs.

In a patient with severe oral impairment, the pharyngeal phase may be elicited by injecting a small barium bolus directly into the pharynx through a soft tube. This may be placed into the pharynx either via the mouth or nose. Such techniques, however, are used only for examination and not for feeding. Patients with uncoordinated, weak or jerky tongue movements commonly cannot correctly position the bolus on the tongue. Accordingly, the tongue cannot displace the bolus posteriorly. There is a strong correlation between an abnormal anterior movement of the hyoid bone and overall abnormal oral and pharyngeal function, as well as defective opening of the PES.

Protection of the airways occurs at four separable anatomically and functionally different sites, i.e. the vocal folds, the supraglottic portion of the laryngeal vestibule, the subepiglottic portion of the laryngeal vestibule and the epiglottis (Curtis and Hudson 1983; Curtis and Sepulveda 1983; Ekberg 1982). The most crucial of these levels is the supraglottic portion of the laryngeal vestibule. Barium in the vestibule commonly extends into the trachea, as the vocal folds offer poor protection of the lower airway (Ekberg and Hilderfors 1985). Radiographic observation of barium penetration into the larynx and trachea is strategic in dysphagia evaluation. Bedside evaluation for aspiration has a low sensitivity. This is partly due to the fact that many of these patients have sensory impairment in the larynx and/or trachea and fail to cough (Splaingard et al. 1988).

Of even more fundamental importance, and basically a prerequisite for airway closure and constrictor activity, is the elevation of the pharynx and larynx. The airways are also protected by a movement of the thyroid cartilage towards the hyoid, and by closure of the laryngeal vestibule. Additional protection is offered by the epiglottis and the vocal folds. Closure of the larynx starts at the vocal folds and progresses in a superior direction in a peristaltic-like manner.

The constrictors have a minor role in the conveyance of a bolus through the pharynx. The tongue-base pressure does not differ significantly between those with and those without retention (Olsson et al. 1997). This finding is important because many research groups advocate normal tongue-base and



a **b**

Fig. 4.1.2a,b. An 80-year-old man with cerebrovascular disease. There is paresis in the right side of the pharynx. This is not seen in lateral projection (**b**) but only in frontal projection (**a**). There is pooling of contrast in the right piriform sinus (*broad arrow*). Small amounts of barium are also seen coating the inside of the laryngeal vestibule down to the false vocal cords

constrictor activity as a prerequisite for a successful outcome of cricopharyngeal myotomy (Buchholz 1995). Tongue-base pressure has been appreciated as the driving force of the bolus, and the tongue-base has been associated with a compensatory function, namely overcoming weak pharyngeal constrictors by increasing tongue-base activity. This tongue driving force propels the bolus while the pharyngeal constrictors stabilise the pharyngeal tube; the tongue then closes the lumen behind the bolus to present retrograde escape (McConnel et al. 1988; Cerenko et al. 1989; McConnel 1988).

In patients with pharyngeal retention the UES does not open as much as in patients without such retention. This reduced opening of the UES is not associated with an increased intrabolus pressure but seen together with decreased laryngeal elevation. This is in agreement with a study in which traction on the UES was found to be the main force leading to the UES opening (Hsieh et al. 1995). The suprahyoid muscles are therefore much more important than other muscles in the pharynx. Also, tongue-base pressure, which is reflected by the intrabolus pressure, was the same in patients with and in those without retention. Thus, pharyngeal shortening could be the most important mechanism in pharyngeal bolus transport (Ergun et al. 1993a, Ergun et al. 1993b).

Such intrabolus pressure was shown to be the same for the two groups in this study.

The pharyngeal phase of swallowing is complex due to the intricate anatomic relationships and the close temporal activation of the more than two dozen muscles that are required to function together to effectively transport the bolus from the mouth to the esophagus. In a classic electromyographic study by Doty and Bosma (1956), temporal activation of the muscles of deglutition was shown. Activation begins in the mylohyoid muscle, and the muscle action is then propelled inferiorly. Early events during the pharyngeal phase of swallowing include activation and sealing of the nasopharynx and contraction of the mylohyoid, hyoglossus, and geniohyoid muscles (i.e. the suprahyoid pharyngeal shorteners). A number of other muscles also contract early, namely, those that effect airway protection, such as the intrinsic and extrinsic muscles of the larynx. Contraction of the superior pharyngeal constrictor, styloglossus, palatoglossus, pterygopharyngeal, palatopharyngeal, stylopharyngeal, salpingopharyngeal, stylohyoid, and posterior digastric muscles then occurs. In terms of bolus transportation, this early stage represents the conveyance of the bolus from the oral cavity into the pharynx. This activity is often described as being achieved by the tongue thrust, although a multitude of muscles are involved (McConnel et al. 1988; Cerenko et al. 1989; McConnel 1988). The pharyngeal constrictors give only stability to the gullet – they do not contribute to bolus transportation in other ways (Dodds 1989). In contrast, the late part of the pharyngeal stage consists of contraction of the thyrohyoid, sternohyoid, sternothyroid, and omohyoid muscles, (i.e. the strap muscles) and also the middle and inferior pharyngeal constrictors. This latter activity is thought to clear the bolus from the pharynx. The late pharyngeal constrictor activity for clearance of the pharynx is seen as a contracting wave traversing inferiorly from the superior pharyngeal constrictor level. This wave is usually best appreciated on the anteroposterior view as an inverted V-shape to the tail of liquid or semi-solid bolus (Dodds 1989). It is impairment of such clearance that is recognised as retention in the valleculae, piriform sinus, or both.

The exact reason why laryngeal elevation leads to retention in patients with manometrically normal pharyngeal constrictor activity is not clear. It is possible that reduced elevation is caused by impaired function of the suprahyoid muscles and other elevators. Such impaired function might lead to abnormal compliance of the wall, against which the constrictors may then act. As retention was not seen in a location immediately cranial to the cricopharyngeal muscle, it is more likely that a defective opening reflects a more profound and wide-spread dysfunction in the pharynx. Defective opening may be the sole result of impaired elevation of the pharynx.

4.1.2.3 The Pharyngoesophageal Segment

The PES is the transition between the pharynx and the esophagus. Between swallows it is kept closed as a sphincter of circular striated muscles. Anatomically it consists of the cricopharyngeal muscle and the inferior portion of the pharyngeal constrictors and the superior portion of the cervical esophagus. The length of the segment is about 3–4 cm where the cricopharyngeal muscle makes up 1–1.5 cm. Closure is the effect of muscle tonicity and the pressure from surrounding tissues (Kahrilas et al. 1988). Normal opening, which is crucial for bolus transport, is achieved by relaxation of muscle tone. However, this probably only accounts for 10%–20% of the total sphincter tonus. More important is the movement of the PES superiorly and anteriorly together with the larynx and hyoid bone. In addition intrabolus pres-

sure created mainly by the tongue-base and constrictor activity also helps to open the PES.

4.1.2.4 The Esophageal Stage

This is comprehensively presented in Sect. 5.1.

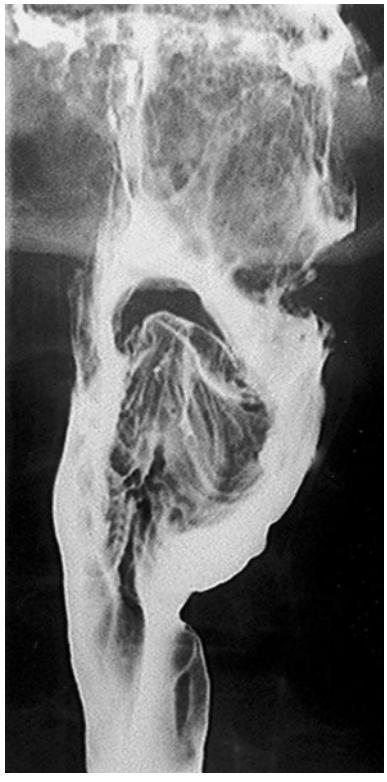
4.1.3 The Abnormal Swallow

In terms of what abnormalities can be expected on the four different anatomic levels, a rule of thumb is that dysfunction is by far the principal abnormality in the oral cavity and pharynx. In the PES, dysfunction and structural abnormalities may co-exist. In the esophagus, structural abnormalities predominate.

4.1.3.1 The Oral Stage

Leaking of barium either anteriorly through the lips, laterally into the buccal pouches or posteriorly into the pharynx is abnormal. Abnormal is also an overly large ingestion in a patient with impaired pharyngeal function and misdirected swallowing. This may indicate impairment of bolus sizing. Impaired lingual movement or jerky uncoordinated movements of the tongue during the preparatory phase of swallowing is also abnormal.

In patients with neurologic diseases, oral dysfunction regularly predominates over pharyngeal dysfunction. Radiographically, this can be appreciated as defective containment, i.e. leakage of barium either anteriorly through the lips, laterally into the buccal pouches or posteriorly – into the pharynx where it potentially may reach the airways if the laryngeal vestibule is not closed. If the patient swallows an abnormally large bolus, this may indicate impairment of bolus sizing. Abnormalities in the oral phase of swallowing, i.e. impaired lingual movement or a soft tissue defect, generally lead to delayed oral transit and clearance of the oral bolus with retention of barium. Premature spill of barium into the pharynx may be accompanied by failed initiation of swallow, aspiration, or both. Impaired oral function is often associated with abnormal pharyngeal swallowing (Fig. 4.1.3). Normally, the oral phase of swallowing undergoes a smooth transition into the pharyngeal phase with vigorous transport of the swallowed bolus into and through the pharynx. In some patients, however, the pharyngeal phase is delayed but otherwise normal.



a

b

Fig. 4.1.3a,b. A 49-year-old man with sudden onset of dysphagia. There is a left-sided weakness of the pharynx. The left side of the pharynx bulges laterally. There is also abnormal opening of the pharyngoesophageal segment, probably due to impaired constrictor strength cranially. The pharyngeal constrictor abnormality is not appreciated in the lateral projection (**a**) but only in the frontal projection (**b**)

4.1.3.2 The Pharyngeal Stage

Abnormal initiation of the pharyngeal stage of swallow is easily appreciated when the bolus is conveyed into the pharynx without the pharynx being elevated and without occurrence of constrictor activity. Again, lack of anterior displacement of the hyoid bone is a conspicuous indicator of serious abnormality. Demonstration of dissociation between the oral and pharyngeal stages depends upon observation of structural displacement. Except for this failure of voluntary elicitation of pharyngeal swallow, the oral as well as the pharyngeal stages of swallow basically appear radiologically normal. In borderline cases, in which the bolus is retained in the valleculae or in the piriform sinuses for 2–5 s, the assessment of normalcy is based upon observation of the bolus.

The ultimate consequences of this dissociation is that the bolus reaches the pharynx when the larynx is still open. Even if the barium does not penetrate into the larynx, the delayed initiation is a potential threat to safe swallow. Even the slightest barium in the vestibule indicates abnormality (Fig. 4.1.4). The wide range of normalcy can be observed during chewing and swallow of a mixture of solids and liquid, especially during talking etc. when the barium and/or solids are brought into the pharynx while chewing goes on without tongue propulsion being elicited. Again, what happens to the bolus is more important than observing wall displacement in this circumstance. It is even more important to recognise that, if pharyngeal swallow is not triggered at the faucial isthmus either by the bolus or the tongue, swallow may be elicited from the vallecula or from the posterior pharyngeal wall.

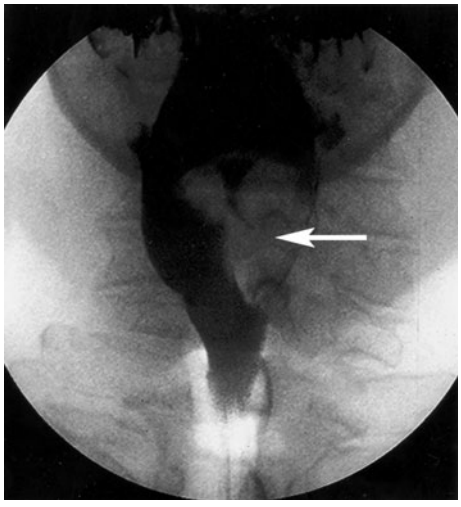


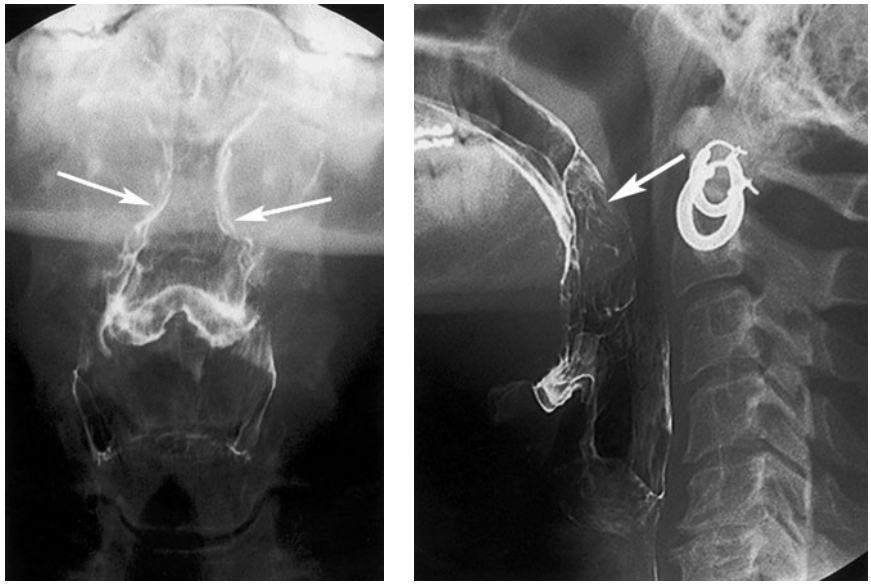
Fig. 4.1.4. Another example of a unilateral pharyngeal constrictor paresis, this time on the right side. The deviation of contrast medium into the right may erroneously give the impression of a tumour on the normal left side (*arrow*). This pseudotumour appearance has led to numerous unnecessary endoscopic examinations

Defective closure of the velopharynx is due to either soft palate dysfunction or defective function in the superior pharyngeal constrictor. Medialward movement of the lateral wall is more extensive than the anterior movement of the posterior constrictor wall. Compensation may be in the form of a Passavant's ridge, a protrusion similar to that seen as a compensatory manoeuvre in speech, in cleft palate persons.

The pharyngeal constrictors play a crucial role in swallowing (Ardran and Kemp 1956). If constrictor muscles are paretic, the pharynx is flaccid and allows an abnormal expansion of the chamber during the compression phase of swallow (Figs. 4.1.3, 4.1.4). Such lack of distensibility may result in impaired transit of bolus from the oral cavity into the esophagus even if the tongue acts normally (Thulin and Welin 1954; Ekberg et al. 1986). This can be seen as a dilated and wide flaccid pharynx but this is a non-specific finding. Action of the pharyngeal constrictors is also crucial for clearing barium from the pharynx. However, there are pitfalls (Fig. 4.1.5). This constrictor activity is best evaluated in the PA view (Fig. 4.1.2). The consequence of defective constrictor activity is residual barium in the pharynx after swallowing. As the middle pharyngeal constrictor is the most commonly involved, retention occurs at the level of the laryngeal inlet and may lead to aspiration after swallow.

In the pharynx, retention of contrast medium may occur and indicate abnormalities in the tongue-base and/or in the pharyngeal constrictors. Any contrast medium that reaches into the laryngeal vestibule and trachea is abnormal (Figs. 4.1.1–4.1.3). This is especially true if the patient does not cough during this event. This indicates desensitisation and is seen in patients with impairment lasting for more than about 2 months. Defective tilting down of the epiglottis is also abnormal and generally seen together with impaired movement of the hyoid bone and larynx. More fundamental abnormalities are impaired elevation of the larynx and pharynx together with the hyoid bone at the initiation of pharyngeal swallow. This is seen in patients who do not elicit the pharyngeal stage of swallow. We have used the beginning of the anterior movement of the hyoid bone as a key event. During the preparatory stage the hyoid and larynx are moving up and down but at the initiation

a b



c d

Fig. 4.1.5a–d. a,b Barium swallow, single contrast full column films. **c, d** Double contrast films. In this 30-year-old woman there are extremely large tonsils (*arrow*). This was seen in both frontal and lateral projection. This may simulate normal or exaggerated function in the constrictor musculature while it in fact is caused by a morphologic abnormality. Such hypertrophic tonsils may cause dysphagia

of the pharyngeal swallow the hyoid starts to move anteriorly (from an elevated position). If that movement is missing the patient does not elicit pharyngeal swallow and is thereby at great risk if fed orally.

4.1.3.2.1 Misdirected Swallowing

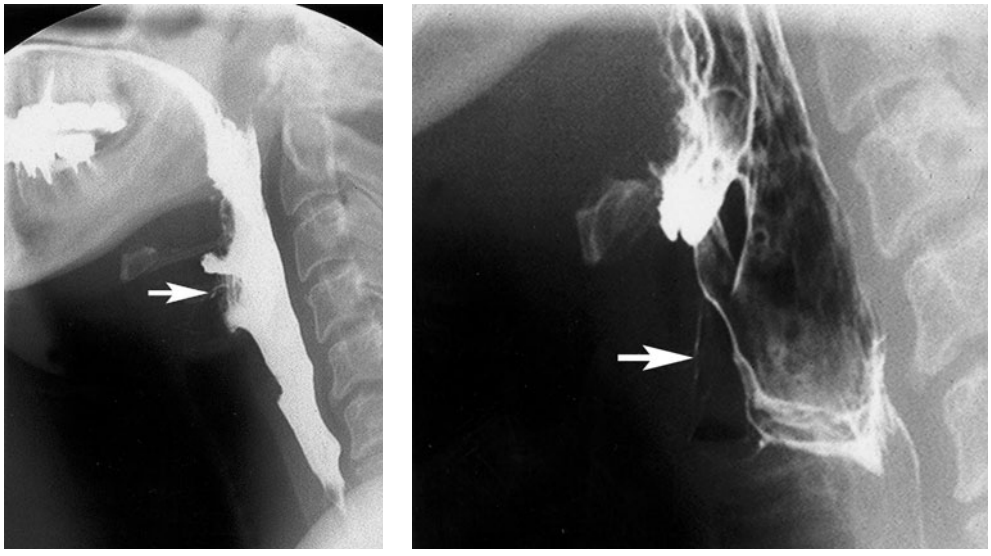
Confusion exists regarding the terminology confined to barium reaching into the airways. *Penetration* has been used either to describe barium reaching into the airways *during* swallow or merely to describe barium reaching only into the laryngeal vestibule and not beyond the vocal folds. *Aspiration* has been used either to describe barium reaching into the airways *after* swallow and usually due to residue in the pharynx or it has been used to described barium reaching beyond the vocal folds. Different use of the terminology has created confusion which is unnecessary. It is much more rational to describe: (1) When

the barium reaches into the airways, namely *before, during* or *after* elicitation of pharyngeal swallow. (2) How far into the airways the barium reaches, namely *into the subepiglottic, supraglottic portion* of the laryngeal vestibule or *beyond the vocal folds*. There is no consensus regarding the terminology and therefore it is warranted to be precise as the implicit meaning of penetration and aspiration is not widely accepted.

Of crucial importance is the co-ordination between the oral and pharyngeal stages of swallowing. The most common cause of misdirected swallowing is dissociation between the oral and pharyngeal stages of swallow. This means that those two compartments are acting normally but the synchronisation between them is impaired. The oral stage includes propulsion of the bolus into the pharynx. The pharyngeal stage of swallowing includes elevation of the hyoid bone, the larynx and the pharynx. It is crucial to observe a conspicuous anterior movement of the hyoid bone as this is an indicator of elicitation of pharyngeal swallow. Any patient who does not show such hyoid bone movement potentially has a severe pharyngeal dysfunction. There should also be an approximation between the larynx and the hyoid bone due to contraction of the thyrohyoid muscles. In normal individuals timing of the oral and pharyngeal stages are tightly co-ordinated and there is usually not a time lag of more than 0.5 s between when the apex of the bolus passes the faucial isthmus and start of the anterior movement of the hyoid bone. In patients with such a tight co-ordination misdirected swallowing is seldom seen. However, the longer the delay the more likely misdirected swallow is to occur. Such incoordination between oral and pharyngeal stage of swallowing is particularly common in the elderly. It is often the sole cause of misdirected swallow (Fig. 4.1.6).

Airway closure and constrictor activity are both important elements of swallow. Absence of elevation of the pharynx and larynx as well as failure of anterior movement of the hyoid bone are crucial elements of abnormal function. These are seen not only in the neurologically impaired patient but also after extensive surgery and/or radiotherapy. During barium swallow, the quality of the cough reflex is readily appreciated and can be defined as to the level where it is elicited, as well as roughly how much and for how long the barium must be in contact with the laryngeal and/or tracheal mucosa. However, for proper quantification of misdirected swallowing to the trachea, scintigraphy is more accurate than barium swallow (Muz et al. 1987).

Discovery of misdirection of the barium into the larynx and/or trachea should not lead to the interruption of the study. On the other hand, there are patients with massive penetration into the trachea in whom a very limited study is sufficient for answering



a **b**

Fig. 4.1.6a,b. This patient complained of an occasional sensation of having a foreign body in the neck. She pointed to the lateral part of her neck. Several swallows were normal. However, small amounts of barium eventually reached into the laryngeal vestibule. This was due to a slight dissociation (1.7 s) between the oral and pharyngeal stage of swallow. This was enough to cause misdirected swallowing into the laryngeal vestibule. The patient then indicated that she had the foreign body sensation in the neck. This was seen both during single-contrast full column film (**a**) (*arrow*) and in double-contrast films (**b**) (*arrow*)

the clinician's immediate questions apropos possible oral feeding. It is important to elucidate the underlying pathophysiology in these patients and a few swallows should be obtained in lateral projection. However, the hazard for acquiring bronchopneumonia secondary to misdirected bolus is probably much less than many would expect (Ekberg and Hilderfors 1985).

According to Kun et al. (1987), elderly patients have misdirected swallowing due to dysfunction in the oral stage or a combination of oral and pharyngeal stage dysfunction. Oral stage dysfunction was due to ingestion of a large volume or rapid acquisition. However, dissociation between the oral and pharyngeal stage was the main finding. It has been shown that thermal stimulation to the faucial isthmus reduces such a dissociation (Logemann 1983; Logemann and Kahrilas 1990). For many years such tactile and thermal stimulation of the faucial isthmus has been a widely used technique for treatment of these patients.

Misdirected swallowing has been considered a major cause of aspiration pneumonia. If major aspiration occurs during barium swallow it is common to put the patient on non-oral feeding. The rationale for such treatment has been questioned (Siebens and Linden 1985). They showed that patients who were kept on non-oral feeding showed more frequent aspiration pneumonia than those who were kept on oral feeding. Their explanation was that saliva contaminated with bacteria in the oral cavity caused the pneumonia. The oral hygiene in these patients is usually low. They conclude that artificial feeding does not seem to be a satisfactory solution for preventing pneumonia in elderly prandial aspirators.

Patients who have defective protection of the airways during swallowing are also at risk for fatal choking episodes. Prandial aspiration most often results in reflexive coughing, gagging and forced exploration. This is often referred to by the patient as "choking". It is usually uncomfortable but brief and usually familiar in nature to the patient. This is in contrast to airway obstruction during oral intake which may be fatal unless the occluding material is removed or displaced. Such food asphyxia is an important cause of accidental death in children (Editorial 1981; Lima 1989). It is estimated that 8000-10,000 adults choke to death each year (Donner and Jones 1985). The development of the Heimlich manoeuvre and its inclusion as part of the basic cardiopulmonary resuscitation training has increased public awareness of the problem in the United States (Heimlich 1985). The cause of near-fatal choking episodes has been studied in 58 individuals (Feinberg and Ekberg 1990). The majority of these patients who had survived a Heimlich manoeuvre due to food impaction showed abnormalities during the barium swallow. The majority aspirated liquid bolus. This was due to bolus leak or dissociation. Some patients also showed defective closure of the laryngeal vestibule. However, a subset of patients (14 of 58) were able to vocalise during the near-fatal choking episode and they demonstrated structural abnormalities of the pharyngoesophageal segment and the esophagus. Therefore, patients who have had a near-fatal choking episode should undergo elective radiologic barium study in order to reveal an underlying cause and prevent new episodes that may prove to be fatal.

4.1.4 Dysfunction of PES

Defective opening of the PES (pharyngoesophageal segment) is a common finding. This may be seen as a posterior indentation of the cricopharyngeal muscle. However, it has been shown that the indentation per se does not impinge on the lumen diameter (Olsson and Ekberg 1995). In fact, the diameter of the PES at the level of the cricopharyngeal muscle is the same in patients with and without that posterior cricopharyngeal bar. Instead the pharynx is dilated above and the cervical esophagus dilated below the cricopharyngeal bar, thereby giving the false impression of lumen narrowing. Manometrically the cricopharyngeal muscle in these patients has been shown to act normally, while there is decreased peak amplitudes in the pharynx above. There is also a small subset of patients who have fibrosis of the PES and the cricopharyngeal muscle but this is rare. The majority of patients who appear to have an abnormal opening of the PES do have normal morphology and function in that particular segment while the impairment in fact is defective elevation of the PES and weakness of the pharyngeal constrictors (Olsson and Ekberg 1995). The most common cause of this in turn is lack of initiation of the pharyngeal stage of swallowing. Therefore, any surgical procedure on the PES is not likely to be beneficial. The main reason being that only a small fraction of the closure of the PES is due to muscle tonicity.

Failure of the PES to open may be seen in neurologic disease but is generally not accompanied by abnormal pharyngeal bolus transport (Curtis et al. 1984, Ekberg 1986a). Failure of the cricopharyngeal muscle to open or elongate may be due to: (1) defective muscle relaxation, (2) defective extensibil-

ity, (3) hypertrophy or hyperplasia. The posterior bar intruding into the barium and created by the cricopharyngeus muscle is seldom an isolated dysfunction. It is commonly associated with abnormal motor function in the segment above (i.e., the inferior pharyngeal constrictor) and/or in the segment below (i.e., the cervical esophageal muscles). Therefore, the cricopharyngeus indentation, though the most conspicuous, is only one aspect of severe motor dysfunction in the adjacent PE segment (Ekberg 1986a). Cervical esophageal webs are also common in dysphagic patients (Figs. 4.1.5, 4.1.7).

Adapted and compensated swallow basically have the same radiologic appearance and, in the majority of patients, it is notoriously difficult to demonstrate. Therefore, a normal radiologic study does not rule out pharyngeal abnormality.

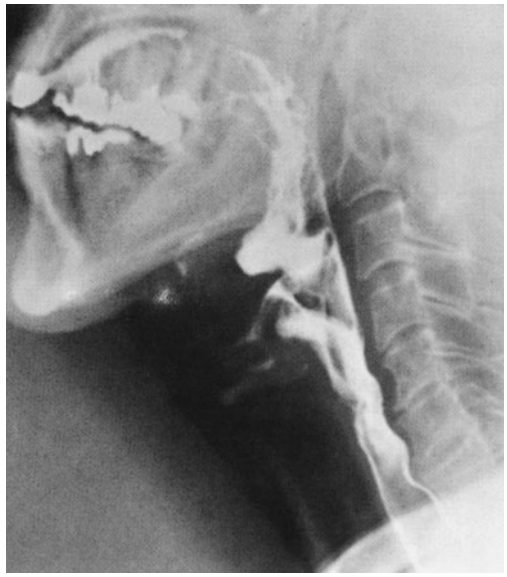


Fig. 4.1.7. A 76-year-old woman 2 weeks after stroke. Lateral radiogram of the pharynx during swallowing. Contrast medium has reached into the pharynx, through the pharyngoesophageal segment and into the cervical esophagus. The epiglottis is in a horizontal position. The hyoid bone is elevated and brought slightly forward. However, contrast medium has reached into the laryngeal vestibule to the level of the sinus Morgagni. No contrast medium is seen in the trachea. The patient did not subjectively experience this misdirected swallow. There was no cough. This indicates chronic aspiration (see also Fig. 4.1.8)

4.1.5 Radiologic Evaluation in Specific Disease Entities

Radiologic evaluation of oral and pharyngeal function during swallowing has a high sensitivity but is non-specific in terms of type and extent of underlying pathology (Figs. 4.1.7–4.1.9). Specific disease is seen to cause pharyngeal dysfunction and dysphagia with varying frequency and the cause of

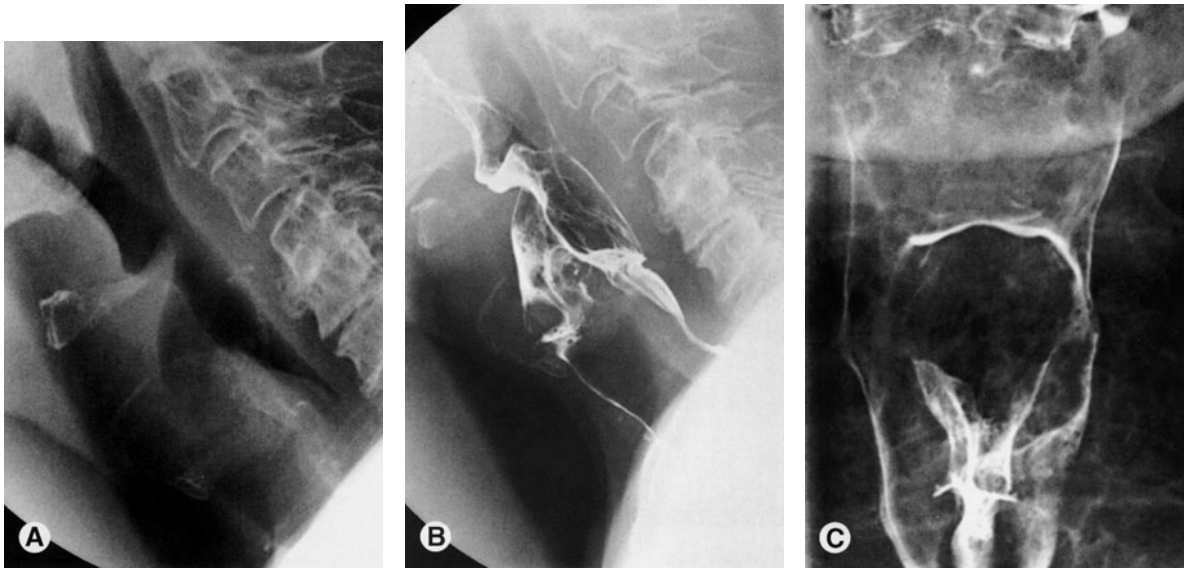


Fig. 4.1.8a–c. A 74-year-old man who had undergone radiotherapy to the neck for laryngeal carcinoma 3 years previously. There was no sign of recurrence but the patient now had difficult swallowing. He had sustained several choking episodes. **a** Lateral radiogram of the pharynx. There is thickening of the prevertebral soft tissue and of the epiglottis. **b** After barium swallow, there is contrast medium in the laryngeal vestibule and in the trachea. Only minor retention in the piriform sinuses. **c** AP view of the pharynx. Minor retention in the effaced valleculae and shallow piriform sinuses. Contrast medium is also seen in the laryngeal vestibule and sinus Morgagni (see also Fig. 4.1.7)

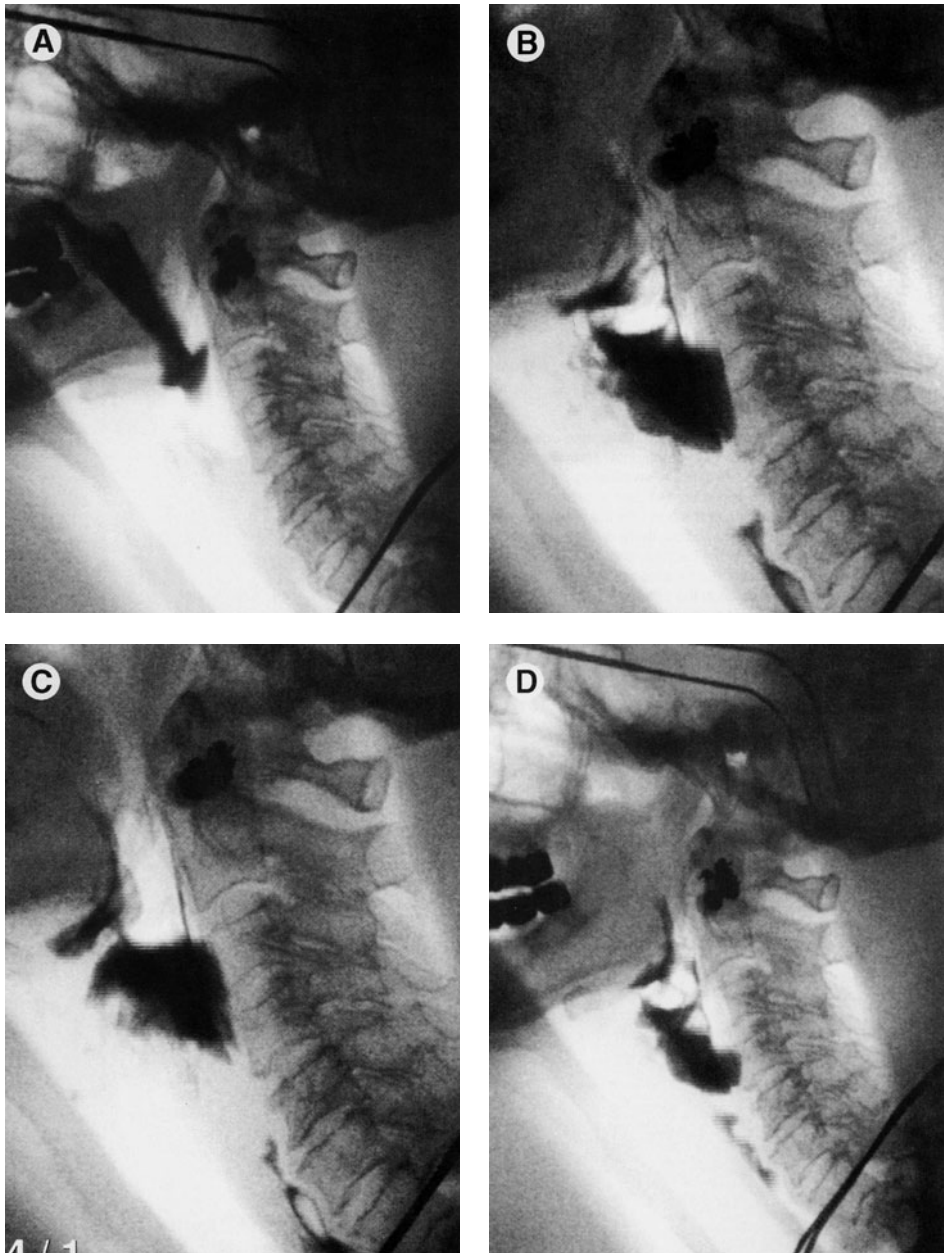


Fig. 4.1.9a–d. A 65-year-old woman with no prior history of stroke. She developed dysphagia 2 years previously. The dysphagia progressed over a couple of weeks. She had difficulty with all kinds of textures and had a very prolonged eating time. The contrast medium is brought into the pharynx (**a**). Pronounced dilatation of the pharynx indicating high compliance secondary to weakness of pharyngeal constrictor musculature (**b**). Eventually contrast medium reached into the laryngeal vestibule but not further down into the airways (**c**). Only very tiny streaks of barium reach through the PE segment during each swallowing attempt (**d**). There was minor anterior movement of the hyoid bone that could indicate that pharyngeal swallow is elicited. However, there is no constrictor activity. The patient has spontaneously learnt to compensate for absence of contractility in the constrictors [From Ekberg and Olsson (1997)]

dysphagia during the disease varies. This has been studied radiologically in cerebrovascular disease (Veis and Logemann 1985; Donner and Silbiger 1966), poliomyelitis (Silbiger et al. 1967; Ardran et al. 1957), amyotrophic lateral sclerosis (Bosma and Brodie 1969a), myasthenia gravis (Murray 1962), myotonic dystrophy (Bosma and Brodie 1969b), Parkinson's disease (Calne et al. 1970; Robbins et al. 1986), brain stem tumour (Kun et al. 1987), and multiple sclerosis (Daly et al. 1962). Even the distinction between upper and lower motor neuron disease, e.g. cortical bulbar tract dysfunction (pseudobulbar palsy) and lower motor neuron disease, i.e. pontomedullary dysfunction (bulbar palsy) is ambiguous. However, the latter patients usually have more widespread pharyngeal paresis and they also lack initiation of pharyngeal swallow. Basically all neurologic impairments lead to the same dysfunction. This is especially so for the "end-stage" dysfunction which tend to lead all types of disease entities down a common pathway with impaired elevation of the larynx and pharynx and impaired anterior movement of the hyoid.

The cause of dysphagia in terms of symptoms and functions often does not match. Deterioration or progression of dysphagia is as a rule compensated. So if we could decompensate swallowing this would reveal progression. The radiologist may intentionally elicit the decompensation by extension of the neck by provision of a large bolus and other stresses (Ekberg 1986b; Buchholz et al. 1985).

4.1.5.1 Cerebrovascular Diseases

The majority of patients with pharyngeal dysfunction are patients who suffer from stroke. The prevalence of stroke in the US has been estimated to approximately 1.6 million persons with 250,000 new cases of stroke each year. Of these stroke patients 30%–50% will develop dysphagia. Strokes that are multiple (Veis and Logemann 1985; Rosenbek et al. 1991; Horner et al. 1991; Splainard et al. 1988), bilateral (Logemann 1983), or localised in the brainstem (Veis and Logemann 1985; Horner et al. 1991; Logemann and Kahrilas 1990; Linden and Siebens 1983) are considered to cause severe swallowing impairment. However, even unilateral cortical or subcortical strokes (Veis and Logemann 1985; Robbins and Levine 1988; Robbins et al. 1993; Gordon et al. 1987; Meadows 1973) can cause swallowing problems. Dysphagia in the stroke population may last for weeks or months but occasionally much longer. Although dysphagia is a subjective symptom, dysfunction of the pharyngeal stage of swallowing can be objectively registered using barium swallow and video recording. This technique is now the gold standard for evaluation of normal and abnormal swallowing.

Oropharyngeal impairment is recognised as a frequent cause of morbidity, disability and costly dependence in stroke patients (Veis and Logemann 1985). Bolus misdirection into the larynx and trachea is perhaps the most significant abnormality that we routinely observe during barium swallow. Especially in the elderly swallowing dysfunction is prevalent. Particularly in those who are hospitalised, institutionalised or well advanced in years. These patients represent some of the most challenging cases that clinicians must deal with on a routine basis. The diagnosis is difficult and so is management. However, this is not because of unique abnormalities in swallowing behaviour or morphodynamics but because of the patients themselves (Veis and Logemann 1985). In fact, swallowing problems may go undetected in these patients because signs and symptoms are vague, subtle or unreported by the patients' caregivers. Critical management decisions regarding dietary alterations, degree of oral intake and institution of artificial feeding often depend on the radiologic assessment of such misdirected swallowing.

Radiologists are important members of multi-disciplinary teams that address swallowing disorders. Diagnosis and treatment of dysphagia depend on videofluorographic deglutition examinations during barium swallow. Patients with cerebrovascular disease, Parkinson's disease and other neurologic conditions, including elderly patients with dementia, need to undergo videofluorographic examination in order to correctly describe functional status and thereby give a platform for rational therapy. Treatment strategies must be founded on objective grounds (see Sect. 4.5).

4.1.6 The Role of the Radiologist in the Design of Therapy

Treatment of oral and pharyngeal dysfunction strives to correct or compensate for damage to specific neuromuscular components of the oral and pharyngeal swallow, such as reduced tongue movement, delay in triggering of the pharyngeal swallow, pharyngeal constrictor paralysis or defective closure of the laryngeal vestibule. For evaluation of such therapy, successive

radiologic evaluations are necessary (see Sect. 4.5). The radiologic examination must: (1) reveal the anatomic and/or physiologic abnormalities causing the patient's dysphagia, and (2) identify those compensatory strategies or therapy techniques which are most effective in improving the efficiency of the patient's oral and pharyngeal swallow. The radiologic examination should start with ingestion and include also the passage of the bolus into the stomach. Following liquid swallows, the patient should be given other food consistencies, particularly pudding consistency materials and food requiring chewing, such as cookies. A variety of foods mixed with barium can be introduced in the radiologic study. The volume of each food or liquid should be measured because the dynamics of normal pharyngeal swallow vary as volume of the bolus increases.

Two types of therapy techniques are used for re-education for oral and pharyngeal dysphagia: (1) "direct" swallowing therapy procedures which passively facilitate swallowing by use of particular foods and liquids; (2) "indirect" therapy techniques designed to compensate for dysfunction by increasing muscle strength, range of motion or co-ordination independent of swallowing. The effectiveness of either of these techniques is assessed by observing the transport of the swallowed bolus during radiography.

References

Ardran GM, Kemp FM (1956) Radiologic investigation of pharyngeal and laryngeal palsy. Acta Radiol Diagn 46: 446–457

Ardran GM, Kemp FM, Wegelius C (1957) Swallowing defects after poliomyelitis. Br J Radiol 30:169–189

Bosma JF, Brodie DR (1969a) Disabilities of the pharynx in amyotrophic lateral sclerosis as demonstrated by cineradiography. Radiology 92:97–103

Bosma JF, Brodie DR (1969b) Cineradiographic demonstration of pharyngeal area myotonia in myotonic dystrophy patients. Radiology 92:104–109

Brühlmann WF (1985) Die röntgenkinematographische Untersuchung von Störungen des Schluckaktes. Huber, Bern

Buchholz DW (1995) Cricopharyngeal myotomy may be effective treatment for selected patients with neurogenic oropharyngeal dysphagia. Dysphagia 10:255–258

Buchholz DW, Bosma JF, Donner MW (1985) Adaptation, compensation, and decompensation of the pharyngeal swallow. Gastrointest Radiol 10:235–239

Calne DB, Shaw DG, Spiers AS, Stern GM (1970) Swallowing in Parkinsonism. Br J Radiol 43:456–457

Cerenko D, McConnel FMS, Jackson RT (1989) Quantitative assessment of pharyngeal bolus driving forces. Otolaryngol Head Neck Surg 100:57–63

Curtis DJ, Cruess DF, Berg T (1984) The cricopharyngeal muscle. A video-recording. AJR 146:497–500

Curtis DJ, Hudson T (1983) Laryngotracheal aspiration: analysis of specific neurmuscular factors. Radiology 149:517–522

Curtis DJ, Sepulveda GV (1983) Epiglottic motion: video recording of muscular dysfunction. Radiology 148:473–477

Daly DD, Code CF, Andersson HA (1962) Disturbances of swallowing and esophageal motility in patients with multiple sclerosis. Neurology 59:250–256

Dodds WJ (1989) The physiology of swallowing. Dysphagia 3:171–178

Donner MW, Jones B (1985) Editorial. Gastrointest Radiol 10: 194–195

Donner MW, Silbiger ML (1966) Cinerafluorographic analysis of pharyngeal swallowing in neuromuscular disorders. Am J Med Sci 251:600–616

Doty RW, Bosma JB (1956) An electromyographic analysis of reflux deglutition. J Neurophysiol 19:44–60

No authors listed (1981) Inhaled foreign bodies (editorial). Br Med J 282:1649–1650

Ekberg O (1982) Defective closure of the laryngeal vestibule during deglutition. Acta Otolaryngol 93:309–317

Ekberg O (1986a) The cricopharyngeus revisited. Br J Radiol 59:875–879

Ekberg O (1986b) Posture of the head and pharyngeal swallow. Acta Radiol Diagn 27:691–696

Ekberg O, Hilderfors H (1985) Defective closure of the laryngeal vestibule: frequency of pulmonary complications. AJR 145:1159–1164

Ekberg O, Lindgren S, Schultz T (1986) Pharyngeal swallowing in patients with paresis of the recurrent nerve. Acta Radiol Diagn 27:697–700

Ekberg O, Olsson R (1997) Operative techniques in otolaryngology. Head Neck Surg 8:153–162

Ekberg O, Wahlgren L (1985) Dysfunction of pharyngeal swallowing: a cineradiographic investigation in 854 dysphagial patients. Acta Radiol Diagn 26:389–395

Ergun GA, Kahrilas PJ, Lin S, Logemann JA, Harig JM (1993a) Shape, volume, and content of the deglutitive pharyngeal chamber imaged by ultrafast computerized tomography. Gastroenterology 105:1396–1403

Ergun GA, Kahrilas PJ, Logemann JA (1993b) Interpretation of pharyngeal manometric recordings: limitations and variability. Dis Esophagus 6:11–16

Feinberg MJ, Ekberg O (1990) Deglutition in near fatal choking episodes: Radiologic evaluation. Radiology 176:637–640

Gordon C, Hewer RL, Wade DT (1987) Dysphagia in acute stroke. Br Med J 295:411–414

Hamlet SL, Stone M, Shawker TH (1988) Posterior tongue grooving in deglutition and speech: preliminary observations. Dysphagia 3:65–68

Hannig C, Hannig A (1987) Stellenwert der Hochfrequenzröntgenkinematographie in der Diagnostik des Pharynx und Ösophagus. Röntgenpraxis 40:358–377

Heimlich JH (1985) A life-saving maneuver to prevent food choking. JAMA 234:398–401

Horner J, Bouyer FG, Alberts MJ, Helms MJ (1991) Dysphagia following brain-stem stroke: clinical correlates and outcome. Arch Neurol 48:1170–1173

Hsieh PY, Brasseur JG, Shaker R, Kern MK, Kahrilas PJ, Ren J (1995) Modeling and timing of UES opening events. Presented at the Dysphagia Research Society meeting, Tysons Corner, McLean, VA, October 26–28, 1995

Jones B, Donner MW (1991) Normal and abnormal swallowing. Imaging in diagnosis and therapy. Springer, Berlin

Kahrilas PJ, Dodds WJ, Dent J, Logemann JA, Shaker R (1988) Upper esophageal sphincter function during deglutition. Gastroenterology 95:52–62

Kun WS, Buchholz D, Kuman AJ, Donner MW, Rosenbaum AE (1987) Magnetic resonance imaging for evaluating neurogenic dysphagia. Dysphagia 2:40–45

Lima JH (1989) Laryngeal foreign bodies in children: a persistent, life-threatening problem. Laryngoscope 99:415–420

Linden P, Siebens A (1983) Dysphagia: predicting laryngeal penetration. Arch Phys Med Rehab 69:637–640

Logemann JA (1983) Evaluation and treatment of swallowing disorders. College-Hill Press, San Diego

Logemann JA, Kahrilas PJ (1990) Relearning to swallow after stroke-application of maneuvers and indirect biofeedback: a case study. Neurology 40:1136–1138

McConnel FMC (1988) Analysis of pressure generation and bolus transit during pharyngeal swallowing. Laryngoscope 98:71–78

McConnel FMS, Cerenko D, Jackson RT, Guffin TN Jr (1988) Timing of major events of pharyngeal swallowing. Arch Otolaryngol Head Neck Surg 114:1413–1418

Meadows JC (1973) Dysphagia in unilateral cerebral lesions. J Neurol Neurosurg Phychiatry 36:853–860

Miller AJ (1986) Neurophysiological basis of swallowing. Dysphagia 1:91–100

Murray JF (1962) Deglutition in myasthenia gravis. Br J Radiol 35:43–52

Muz J, Mathog RM, Miller PR, Rosen R, Borrero G (1987) Detection and quantification of laryngotracheopulmonary aspiration with scintigraphy. Laryngoscope 97:1180–1185

Olsson R, Castell J, Johnston B, Ekberg O, Castell DO (1997) Combined videomanometric identification of abnormalities related to pharyngeal retention. Acad Radiol 4:349–354

Olsson R, Ekberg O (1995) Videomanometry of the pharynx in dysphagic patients with a posterior cricopharyngeal indentation. Acad Radiol 2:597–601

Pokieser P, Schober W, Schima W (1995) Videokinematographie des Schluckaktes–-Indikation, Methodik und Befundung. Radiologe 35:703–711

Robbins J, Levine RL (1988) Swallowing after unilateral stroke of the cerebral cortex: preliminary experience. Dysphagia 3: 11–17

Robbins J, Levine RL, Maser A, Rosenbek JC, Kempster GB (1993) Swallowing after unilateral stroke of the cerebral cortex. Arch Phys Med Rehab 74:1295–1300

Robbins JA, Logemann JA, Kirshner HS (1986) Swallowing and speech production in Parkinson´s disease. Ann Neurol 19:283–287

Rosenbek JC, Robbins J, Fishback B, Levine RL (1991) The effects of thermal application on dysphagia after stroke. J Speech Hear Res 34:1257–1268

Siebens AA, Linden P (1985) Dynamic imaging for swallowing re-education. Gastrointest Radiol 10:251–253

Silbiger M, Pikielney R, Donner MW (1967) Neuromuscular disorders affecting the pharynx. Invest Radiol 2:442–448

Splainard ML, Hutchins B, Sulton LD, Chaudhuri G (1988) Aspiration in rehabilitation patients: videofluoroscopic versus bedside clinical assessment. Arch Phys Med Rehab 69:637–640

Thulin A, Welin S (1954) Radiographic findings in unilateral hypopharyngeal paralysis. Acta Otolaryngol Suppl 116: 288–293

Veis SL, Logemann JA (1985) Swallowing disorders in persons with cerebrovascular accident. Arch Phys Med Rehabil 66: 372–375

4.2 Pharyngeal Morphology

Stephen E. Rubesin

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4.2.1 Introduction

There is no dividing line between morphologic and functional disorders of the pharynx. Structural abnormalities frequently cause abnormal pharyngeal motility. Motility disorders often are manifested by abnormal pharyngeal structure. Therefore, examination of the pharynx requires a dynamic examination of motility in combination with static images of morphology. The radiologist tailors the dynamic (videofluoroscopy) and morphologic (spot image) examination to the patient's clinical history, symptoms and the initial fluoroscopic findings (Rubesin and Glick 1988; Rubesin and Stiles 1997). This chapter will discuss morphologic disorders of the pharynx.

S. E. Rubesin, MD
Professor of Radiology, University of Pennsylvania School of Medicine, Department of Radiology, MRI Building 1-08, University of Pennsylvania Medical Center, 3400 Spruce St., Philadelphia, PA 19104-4283 USA

4.2.2 Surface Anatomy Pertinent to Roentgen Interpretation

The principles of interpreting roentgen images of the pharynx are the same as in the evaluation of the remainder of the gastrointestinal tract (Rubesin and Laufer 1991). The shape of the pharynx is determined by the tongue, laryngeal cartilages (epiglottic, thyroid, cricoid, and arytenoid cartilages), the pharyngeal musculature, and the supporting skeleton. The normal surface of distended pharyngeal mucosa is smooth (Fig. 4.2.1), except in the regions of the palatine and lingual tonsils. The normal surface of the palatine and lingual tonsils is slightly nodular due to the underlying lymphoid tissue (Figs. 4.2.2 and 4.2.3) (Grommet et al. 1982). When the pharynx is slightly collapsed, barium-coated striations may be seen (Fig. 4.2.4a) (Rubesin et al. 1987a). These lines reflect the underlying vertically-oriented inner longitudinal muscle layer of the pharynx: the fibers and aponeurosis of the salpingopharyngeal, stylopharyngeal and palatopharyngeal muscles (Fig. 4.2.4b). Transversely-oriented lines are seen in the lower hypopharynx and reflect redundant epithelium overlying the muscular processes of the arytenoid cartilages and cricoid cartilage. Thus, any nodularity or focal barium collection that disrupts the smooth mucosal surface of the lateral or posterior pharyngeal wall is suspicious for an inflammatory or neoplastic process.

During pharyngography, the mucosa behind the cricoid cartilage appears undulating (Fig. 4.2.5a). Although this prominent post-cricoid mucosa was thought reflect a venous plexus (Pitman and Fraser 1965), redundant squamous epithelium and submucosa explain the radiographic findings (Fig. 4.2.5b) (Rubesin et al. 1987a). As long as the post-cricoid mucosa changes size and shape during swallowing, a post-cricoid tumor need not be considered.

Fig. 4.2.1. Arcuate lines in hypopharynx. After the patient aspirated a small amount of barium, a spot radiograph of pharynx shows the relationship of the barium-coated larynx (right true vocal cord identified with t) to the hypopharynx. The larynx acts as an extrinsic mass impression upon the lower hypopharynx. Arcuate lines (*arrows*) reflect redundant mucosa in the hypopharynx. As long as the arcuate lines are smooth and thin, no tumor or inflammatory process should be considered (Reproduced with permission from Rubesin SE, Glick SN (1988) The tailored double-contrast pharyngogram. Crit Rev Diagn Imaging 28:133–179, Fig. 11a)

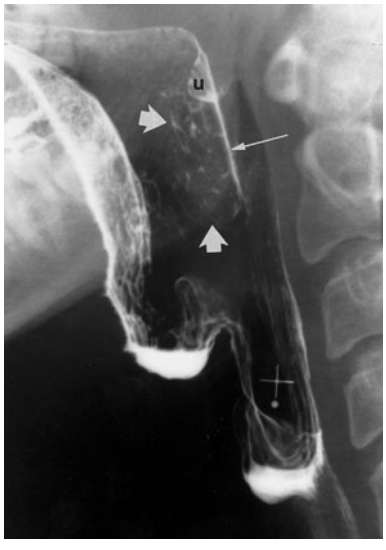


Fig. 4.2.3. Normal mucosal surface of the palatine tonsils. Spot radiograph of pharynx shows barium filling the interstices of the normal lymphoid tissue of the palatine tonsil (*thick arrows*). Also note the uvular tip (*u*) of the soft palate and the palatopharyngeal fold (*long arrow*) (Reproduced with permission from Rubesin SE, Jones B, Donner MW (1987) Contrast pharyngography: the importance of phonation. AJR 148:269–272, Fig. 2b)

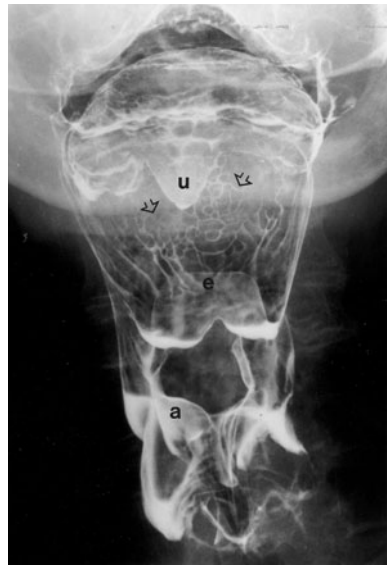


Fig. 4.2.2. Mucosal surface base of tongue. Spot radiograph of pharynx demonstrates a reticular pattern (representative area identified by open arrows) reflecting barium filling the interstices of the fine nodular surface of the base of the tongue. The reticular surface reflects the underlying lingual tonsil. Disruption of the normal, relatively flat reticular surface of the tongue is abnormal. Also identified are the uvula (*u*), epiglottic tip (*e*). The mucosa overlying the muscular processes of the arytenoids (right arytenoid identified by a) is elevated due to radiation-induced edema. The left hypopharynx, the site of the radiated cancer, is contracted

4.2.3 Pouches and Diverticula

Pouches are transient protrusions at sites of anatomic weakness of the pharyngeal wall, while diverticula are persistent protrusions of pharyngeal mucosa and submucosa at these same sites (Perrot 1962).

4.2.3.1 Lateral Pharyngeal Pouches and Diverticula

The lateral pharyngeal wall most frequently protrudes in the region of the thyrohyoid membrane (Fig. 4.2.6), an area bounded superiorly by the greater cornu of the hyoid bone, inferiorly by the ala of the thyroid cartilage, anteriorly by the posterior border of the thyrohyoid muscle and posteriorly by the superior cornu of the thyroid cartilage and the insertion of the stylopharyngeal muscle (Fig. 4.2.7) (Dubrul 1980). The unsupported portion of thyrohyoid membrane is perforated by the superior laryngeal artery and vein and the internal laryngeal branch of the superior laryngeal nerve (Pernkopf 1989). It is not known what happens to this space during swallowing, when

Fig. 4.2.4a,b. Lines of the pharynx. **a** Spot radiograph shows vertically oriented striations (*arrows*) in the lateral and posterior surface of the hypopharynx. These lines reflect the underlying longitudinal muscle layer. **b** Low-power photomicrograph of the lateral pharyngeal wall shows close apposition of the squamous epithelium (*short arrow*) to the longitudinal muscle layer (*long arrowhead*). Only a thin tunica propria (*long straight arrow*) separates the squamous epithelium from the longitudinal muscle layer. The constrictor muscle layer is identified by a curved arrow (a and b reproduced with permission from Rubesin SE, Glick SN (1988) The tailored double-contrast pharyngogram. Crit Rev Diagn Imaging 28:133–237, Fig. 5a and 6, respectively)

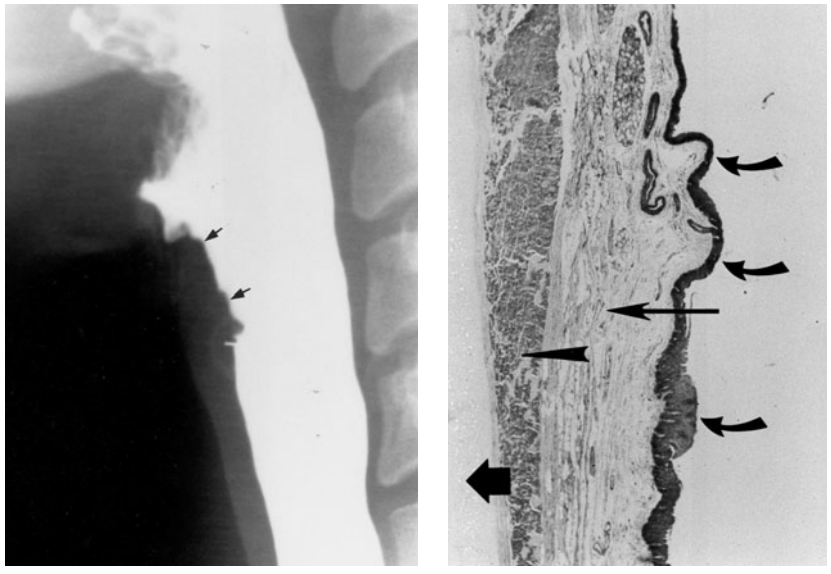
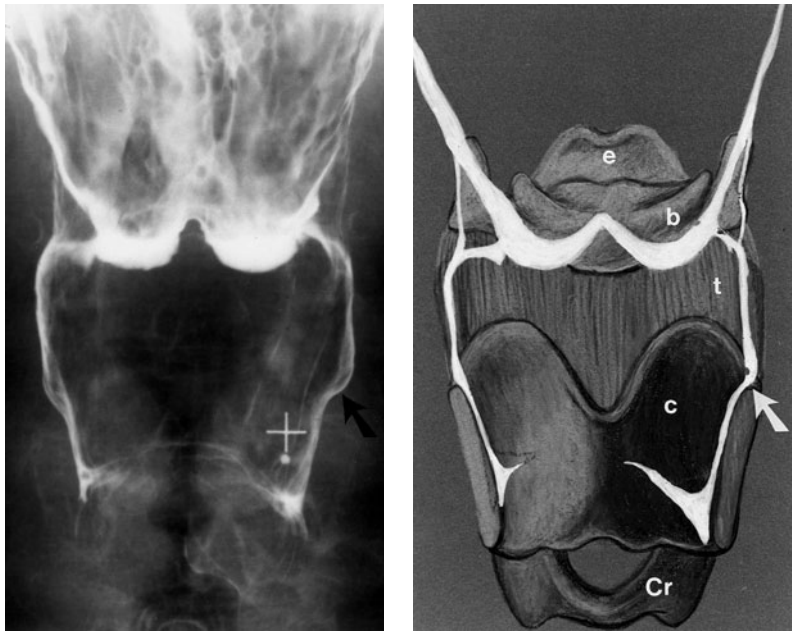
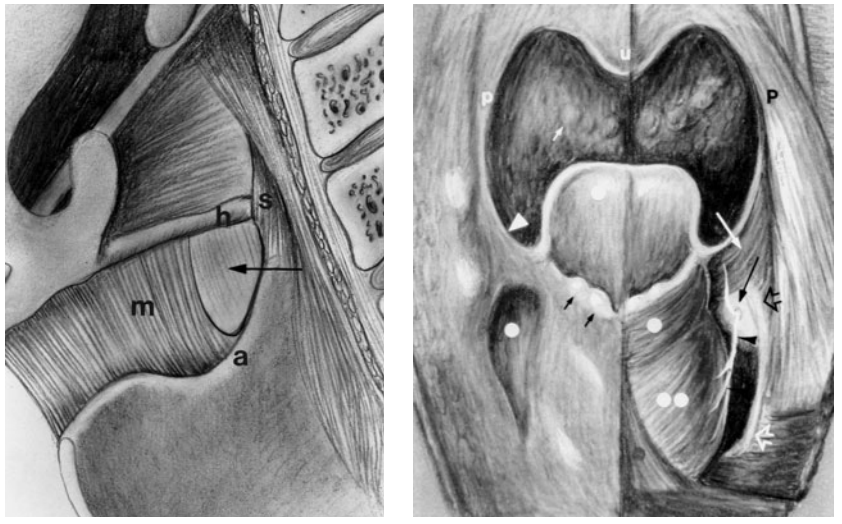


Fig. 4.2.5a,b. Post-cricoid mucosa. **a** Spot radiograph of the pharynx obtained while the bolus pass through the pharyngoesophageal segment shows undulating mucosa (*arrows*) just behind the cricoid cartilage. **b** Low-power photomicrograph obtained at the level of the cricoid cartilage (*thick arrow*) shows a sinuous squamous epithelium (*curved arrows*) corresponding to the undulating mucosa seen in **a**. The submucosa (*long arrow*) is very thick at this level, reflecting the need for this area to move easily. Compare the thickness of the submucosa here to the tunica propria in 4b. The cricoarytenoid muscle (*arrowhead*) lies posterior to the cricoid cartilage (A reproduced with permission from Rubesin SE (2000) Pharynx. In Levine MS, Rubesin SE, Laufer I (eds) Double contrast gastrointestinal radiology. Philadelphia, WB Saunders, pp. 60–89, Fig. 47a; Fig. b reproduced with permission from Rubesin SE et al. (1988) The tailored double-contrast pharyngogram. Crit Rev Diagn Imaging 28:133–237, Fig. 8)



a b

Fig. 4.2.6a,b. The thyrohyoid membrane. **a** Spot radiograph of the pharynx shows focal indentation of the contour of the lateral hypopharyngeal wall (*arrow*). This marks the transition between the hypopharynx that lies above the ala of the thyroid cartilage and the portion of hypopharynx confined by the thyroid cartilage. **b** Corresponding line drawing demonstrates the point where the anterolateral hypopharyngeal wall becomes confined by the thyroid cartilage (*arrow*). The thyrohyoid membrane (*t*) bridges the space between the hyoid bone (*b*) and the thyroid cartilage (*c*). Also note the epiglottic cartilage (*e*) and cricoid cartilage (*Cr*) (a and b reproduced with permission from Rubesin SE et al. (1987) Lines of the pharynx. RadioGraphics 7:217–237, Fig. 10a and 10b)



a b

Fig. 4.2.7a,b. Location of lateral pharyngeal pouches. **a** Line drawing performed in the lateral view shows the location of the unsupported portion of the thyrohyoid membrane (*arrow*): posterior to the thyrohyoid muscle (*m*) and membrane, anterior to the superior cornu of the thyroid cartilage and inserting fibers of the stylopharyngeus muscle (*s*), inferior to the hyoid bone (*h*), and superior to the ala of the thyroid cartilage (*a*). **b** Dissection of pharynx viewed from behind shows the unsupported portion of the thyrohyoid membrane (*black arrow*) and the internal branch of the superior laryngeal nerve (*black arrowheads*). The thyroid cartilage is identified by open arrows. Also identified are the palatopharyngeal fold (*white P*) and its corresponding palatopharyngeus (*black P*), the uvula (*u*), the circumvallate papillae (*tiny white arrow*), the left pharyngoepiglottic fold (*arrowhead*), the left piriform sinus (*one white dot on the left*), the mucosa overlying the cuneiform and corniculate cartilages (*tiny black arrows*), the right arytenoid muscle (*one white dot on the right*) and the cricoarytenoid muscle (*two white dots on the right*) (a and b reproduced with permission from Rubesin SE et al. (1987) Lines of the pharynx. RadioGraphics 7:217–237, Figs. 8b, 11c)

the hyoid bone and thyroid cartilage are brought together by contraction of the thyrohyoid muscle.

Lateral pharyngeal pouches are extremely common, the incidence increasing with patient age. Only a small percentage (about 5%) of patients with lateral pharyngeal pouches are symptomatic, complaining of dysphagia, choking, or regurgitation of undigested food (Curtis et al. 1988; Lindbichler et al. 1998).

During swallowing, in the frontal view, lateral pharyngeal pouches are manifested as transient sac-like protrusions (Fig. 4.2.8) of the lateral wall above the notch in the lateral pharyngeal wall that denotes the junction of the ala of the thyroid cartilage and the thyrohyoid membrane. The neck of the sac changes size during swallowing when the thyroid cartilage is raised to appose the hyoid bone. On the lateral view, pouches are manifested as barium-filled sacs on the anterior hypopharyngeal wall just below the hyoid bone at the level of the valleculae. Barium retained in the pouches is spilled into the lateral swallowing channels just after the bolus passes.

Lateral pharyngeal diverticula are persistent protrusions at the level of the thyrohyoid membrane. These diverticula are barium-filled sacs which usually fill via a narrow neck. In contrast to lateral pharyngeal pouches, lateral pharyngeal diverticula are often unilateral (Fig. 4.2.9).

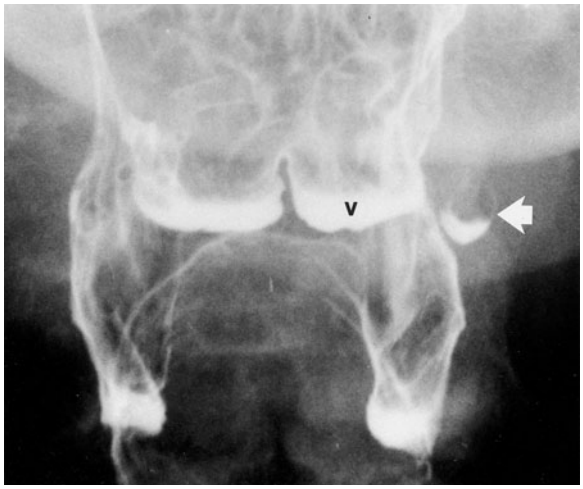
4.2.3.2 Branchial Cleft Cysts and Fistulas

Branchial clefts are grooves of ectodermal origin that develop on both sides of the neck in a 4-week-old embryo (Hyams et al. 1988; Maran and Buchanan 1978). Branchial pouches are four pharyngeal outpouchings of endodermal origin that meet the branchial clefts. The first branchial cleft forms the external auditory meatus. The second branchial cleft forms the middle ear, eustachian tube and floor of the tonsillar fossa. The third and fourth branchial pouches form the piriform sinuses. Persistence of either a branchial cleft or pouch may result in a sinus tract or cyst.

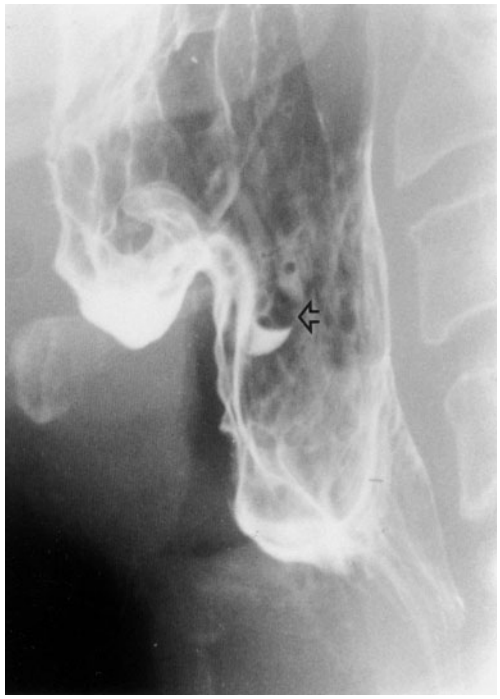
The most common branchial vestige is a cyst arising from the second branchial cleft. Small second branchial cleft cysts lie anterior to the sternocleidomastoid muscle. Larger cysts may extend below the sternocleidomastoid muscle between the internal and external carotid arteries. These cysts only rarely communicate with the pharynx (Bachman et al. 1968).

Branchial pouch sinuses end blindly in the soft tissue of the neck. Branchial pouch fistulas extend to the skin. Branchial pouch sinuses and fistulas arise from the tonsillar fossa (second pouch), the upper anterolateral wall of the piriform sinus (third

Fig. 4.2.8a,b. Lateral pharyngeal pouches. **a** Spot radiograph obtained during drinking shows a small right (*small white arrow*) and larger left (*large white arrow*) lateral pharyngeal pouch. Note the relationship with the hyoid bone (*h*). The epiglottis (*small black arrows*) is tilting asymmetrically. Note the height of the right lateral pharyngeal pouch (*double arrow*) while the patient drinks the bolus. **b** Spot radiograph obtained just after the swallow in A shows that the pharynx has descended to its normal resting position. The notch identifying the superior border of the thyroid cartilage is identified (*white arrow*). Note the difference in height of the thyrohyoid membrane during swallowing (*double arrow* in **a**) and at rest (*double arrow* in **b**)



a



b

Fig. 4.2.9a,b. Lateral pharyngeal diverticulum. **a** Spot radiograph obtained with patient in frontal position shows a 5 mm ovoid sac (*arrow*), partially filled with barium. Note that the diverticulum protruding from the left lateral upper hypopharyngeal wall and is about at the level of the valleculae (*left vallecula identified with v*). **b** Spot radiograph obtained with patient in lateral position shows that the ovoid diverticulum (*arrow*) arises from the anterior portion of the lateral hypopharyngeal wall

pouches) and the lower anterolateral wall of the piriform sinus (fourth pouches). Although most of these sinuses and pouches are present at birth, sinus tracts are occasionally detected for the first time in adults (Fig. 4.2.10).

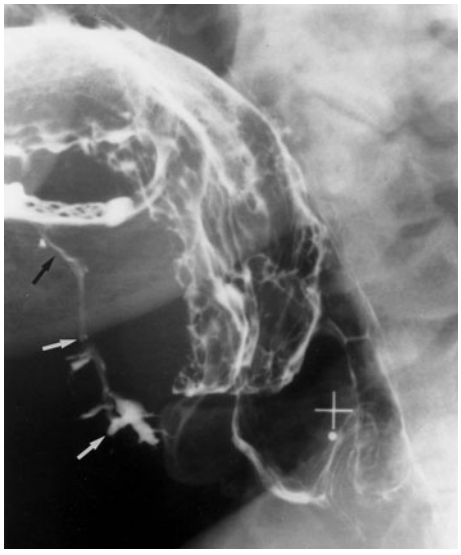


Fig. 4.2.10. Second branchial pouch sinus. Spot radiograph with patient in steep right posterior oblique position shows an irregular barium filled track (*arrows*) arising from the region of the right palatine fossa (Reproduced with permission from Rubesin SE, Glick SN (1988) The tailored double-contrast pharyngogram. Crit Rev Diagn Imaging 28:133–179, Fig. 23b)

4.2.3.3 Zenker's Diverticulum

Zenker's diverticulum (posterior pharyngeal diverticulum) is an acquired mucosal herniation through Killian's dehiscence, a gap in the region of the cricopharyngeal muscle, found in about one-third of individuals at autopsy (Zaino et al. 1970). There is considerable variation in the anatomy of the thyropharyngeus and the cricopharyngeus. Thus, Killian's dehiscence has been described as arising either between the thyropharyngeus and the cricopharyngeus or between the oblique and transverse fibers of the cricopharyngeus (Perrot 1962; Zaino 1967; Zaino et al. 1970).

The relationship between Zenker's diverticulum and the function of the cricopharyngeus is not known. In some studies, upper esophageal sphincter (UES) pressure is normal (there is no spasm), the muscle relaxes completely during swallowing (there is no achalasia) and there is normal coordination between pharyngeal contraction and UES relaxation (Knuff et al. 1982; Frieling et al. 1988). Other studies have suggested that there is either abnormal relaxation of the UES or incoordination of pharyngeal contraction. It is also not known whether chronic gastroesophageal reflux predisposes to the development of Zenker's diverticulum. Clearly, between 65 and 95% of patients with Zenker's diverticulum have gastro-

esophageal reflux (Smiley et al. 1970; Delahunty et al. 1971; Rubesin and Levine 2001)

Zenker's diverticulum is usually first detected in elderly patients who complain of dysphagia, halitosis, choking, hoarseness, or regurgitation of undigested food. Zenker's diverticulum is not infrequently found in asymptomatic individuals or patients being studied for symptoms of gastroesophageal reflux disease. Change in character of dysphagia or bloody discharge in a patient with a known Zenker's diverticulum suggests development of a complication such as ulceration, fistula formation or carcinoma (Nanson 1976; Shirazi et al. 1977).

Radiographically, in the frontal view, Zenker's diverticulum appears as a barium-filled sac midline below the tips of the piriform sinuses (Figs. 4.2.11 and 4.2.12). In the lateral view, Zenker's diverticulum appears as a barium-filled sac posterior to a prominent pharyngoesophageal segment and the upper cervical esophagus (see Fig. 4.2.12). During swallowing, Zenker's diverticulum appears as a protrusion of the lower hypopharyngeal wall posterior to the expected luminal contour, the neck of the diverticulum originating above a "prominent" pharyngoesophageal segment. The opening of the diverticulum may be very large during swallowing, almost 2 cm in height (Fig. 4.2.13). After the swallow has passed, barium regurgitates back into the hypopharynx, in rare cases, resulting in overflow aspiration. Any irregularity of the contour of the diverticulum suggests development of an ulcer or carcinoma (Wychulis et al. 1969).

Zenker's diverticulum should not be confused with a "pseudo-Zenker's diverticulum", barium trapped above a cricopharyngeus that has either opened incompletely or closed early (Fig. 4.2.14). Some pseudo-Zenker's diverticula are pouches arising at Killian's dehiscence. It is not known whether a Zenker's diverticulum can develop from a pseudo-Zenker's diverticulum. This author believes that many pseudo-Zenker's diverticula result from cricopharyngeal response to gastroesophageal reflux (Brady et al. 1995).

4.2.3.4 Killian-Jamieson Pouches and Diverticula

Killian-Jamieson diverticula protrude through the Killian-Jamieson space, a gap in the muscle of the proximal cervical esophagus. This gap is bounded superiorly by the inferior margin of the cricopharyngeal muscle, anteriorly by the inferior margin of the cricoid cartilage and inferomedially by the suspensory ligament of the esophagus just below its origin on the posterior lamina of the cricoid

a b

Fig. 4.2.11a,b. Small Zenker's diverticulum. **a** Spot radiograph with patient in frontal position shows a 5 mm ovoid barium-filled sac (*arrow*) midline below the tips of the piriform sinuses. **b** Spot radiograph with patient in lateral position shows a 4 mm barium-filled sac (*arrow*) posterior to the expected lumen of the pharyngoesophageal segment. Note that this tiny diverticulum persists after swallowing but does not extend posterior to the pharyngoesophageal segment

Fig. 4.2.12a,b. Moderately large Zenker's diverticulum. **a** Spot radiograph with patient in frontal position shows a 3 cm barium-filled sac (*arrow*) midline below the tips of the piriform sinuses. **b** Spot radiograph with patient in lateral position shows a relatively flat, but long barium-filled sac (*large arrows*) posterior to the pharyngoesophageal segment and upper cervical esophagus (*small arrow*)

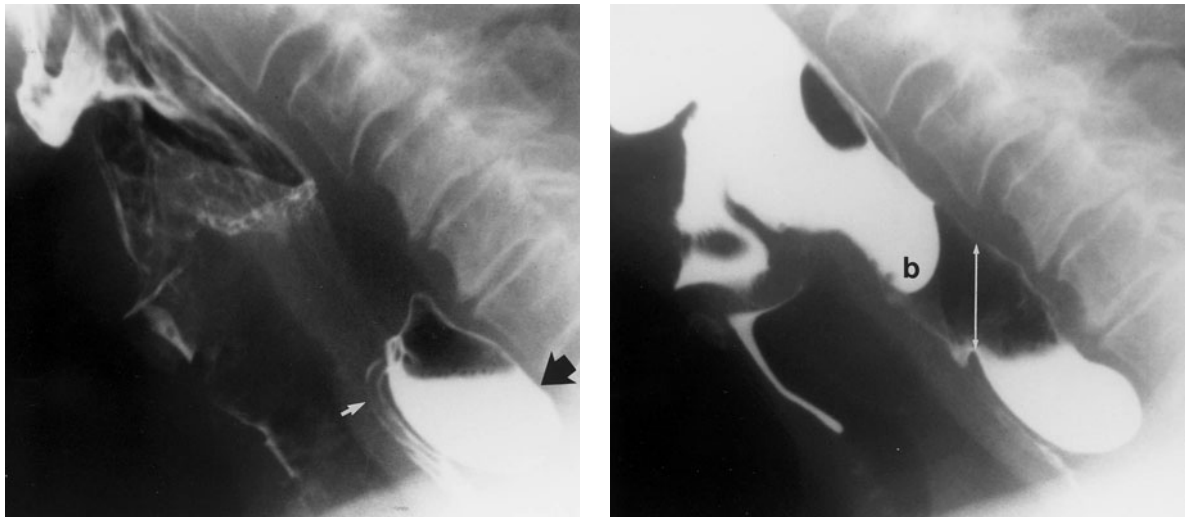


Fig. 4.2.13a,b. Opening of a moderately large Zenker's diverticulum. **a** Spot radiograph obtained at rest with patient in lateral position shows a 2 × 3 cm sac (*large arrow*) posterior to the pharyngoesophageal segment (*small arrow*) and upper cervical esophagus. **b** Spot radiograph obtained just as the bolus (*b*) approaches the pharyngoesophageal segment shows that the opening (*double arrow*) of the Zenker's diverticulum is very tall, at least the height of one vertebral body. Barium entering the laryngeal vestibule was due to abnormal timing between the oral and pharyngeal phase of swallowing (**b** reproduced with permission from Rubesin SE (1991) Pharyngeal dysfunction. In Gore RM (ed) Categorical course on gastrointestinal radiology. Reston, VA, American College of Radiology, 1–9, Fig. 5c)

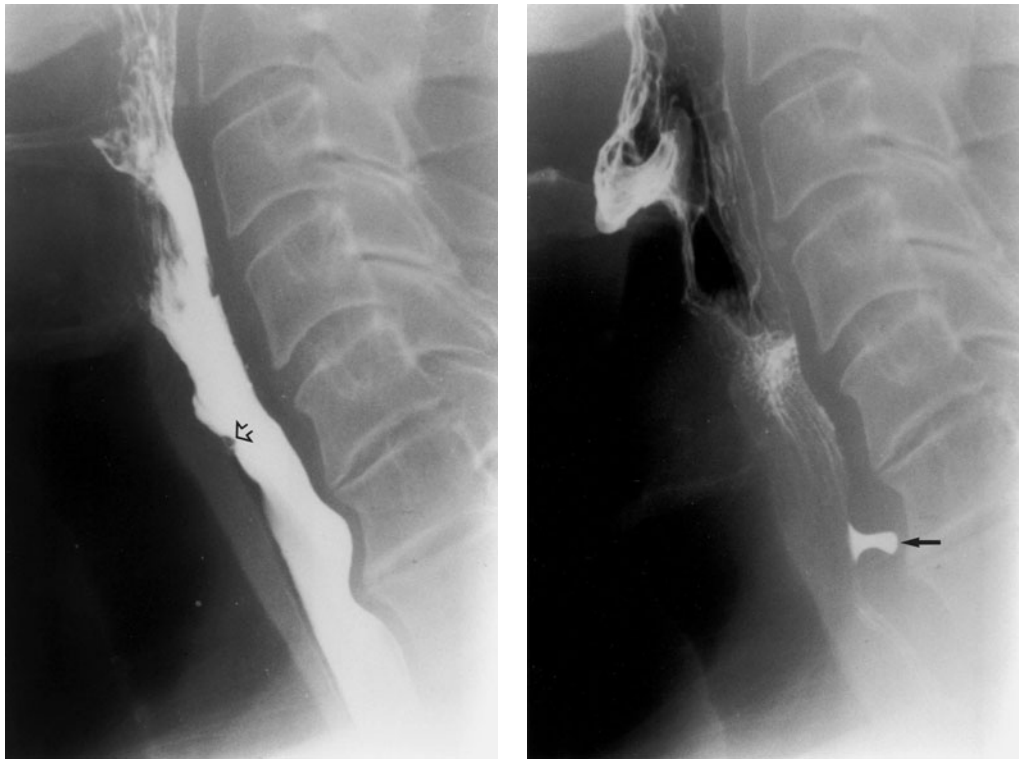


Fig. 4.2.14a,b. Pseudo-Zenker's diverticulum. **a** Spot image obtained during swallowing shows no evidence of a diverticulum at the level of the cricoid cartilage, as identified by redundant post-cricoid mucosa (*open arrow*). **b** Spot image obtained just after the swallow has passed shows barium trapped (*arrow*) above a cricopharyngeal bar that has closed early. Seconds later the pseudo-Zenker's diverticulum disappeared when the collection of barium entered the cervical esophagus

cartilage (Killian 1908). These diverticula are also known as "proximal lateral cervical esophageal diverticula" or "lateral diverticula from the pharyngoesophageal junction area" (Ekberg and Nylander 1983a). Patients with Killian-Jamieson diverticula are usually asymptomatic or have symptoms due to abnormal pharyngeal motility (Rubesin and Levine 2001).

During pharyngography, the opening of the Killian-Jamieson diverticulum lies just below the cricopharyngeus (Fig. 4.2.15). The opening of the sac changes size and shape with elevation of the cervical esophagus during swallowing (see Fig. 4.2.15). The sac of the diverticulum lies lateral to the proximal cervical esophagus on frontal views and overlaps the cervical esophagus on lateral views. Killian-Jamieson diverticula are more frequently unilateral than bilateral and are usually left-sided (Fig. 4.216) (Rubesin and Levine 2001). Bilateral diverticula are seen in about one-quarter of patients. Killian-Jamieson diverticula are smaller than Zenker's diverticula, averaging about 1.4 cm (Rubesin and Levine 2001). Regurgitation of barium from the sac into the hypopharynx is uncommon because regurgitation is prevented by the cricopharyngeus muscle. Occasionally, Killian-Jamieson diverticula and a Zenker's diverticulum are seen in the same patient (Fig. 4.2.17).

Pouches are also frequently detected at the Killian-Jamieson space. These pouches may be related to early closure of the upper cervical esophagus, a finding associated with gastroesophageal reflux. On the frontal view, pouches appear as shallow, broad-based protrusions of the lateral proximal cervical esophageal wall; these pouches are effaced during swallowing (Ekberg and Nylander 1983a).

4.2.4 Inflammatory and Other Lesions

4.2.4.1 Lymphoid Hyperplasia

The normal surface of the base of the tongue has a reticular pattern created by the underlying lingual tonsil, an aggregate of 30-100 follicles extending from

Fig. 4.2.15a–d. Killian-Jamieson diverticula. **a** Spot radiograph obtained at the end of the swallow shows a 1.3 cm diverticulum (*thick arrow*) arising from the left lateral wall just below the level of the cricopharyngeus. The neck (*double arrow*) of the diverticulum is broad during swallowing. **b** Spot radiograph obtained just after the bolus passed shows a narrower neck (*double arrow*) of the diverticulum. **c** Spot radiograph obtained with the patient in the lateral position shows a 1.3 cm diverticulum (*large arrow*) below the level of the cricopharyngeus. Part of the diverticulum lies anterior to the expected course of the pharyngoesophageal segment and upper cervical esophagus (*small arrow*). Barium in the laryngeal vestibule and proximal trachea was related to a pharyngeal motor disorder. **d** Spot radiograph obtained during swallowing demonstrates that part of the diverticulum (*white arrow*) lies anterior to the pharyngoesophageal segment. The presence of a prominent cricopharyngeus (*black arrow*) demonstrates that the diverticulum lies below the cricopharyngeus (a, b, c, and d reproduced with permission from Rubesin SE, Levine MS (2001) Killian-Jamieson diverticula: radiographic findings in 16 patients. AJR 177:85–89, Fig. 1)

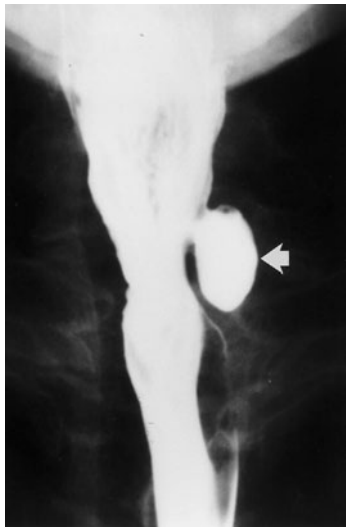
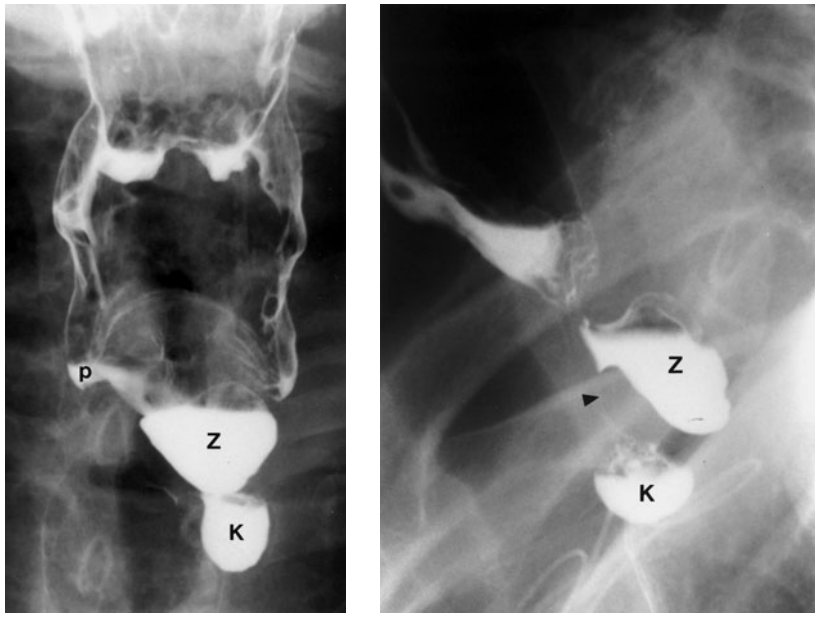


Fig. 4.2.16. Killian-Jamieson diverticulum. Spot radiograph obtained during swallowing shows a 1.5 cm barium-filled sac (*arrow*) arising from the left lateral wall near the pharyngoesophageal segment. After swallowing the orifice of the diverticulum was shown to be below the cricopharyngeus (Reproduced with permission from Rubesin SE, Levine MS (2001) Killian-Jamieson diverticula: radiographic findings in 16 patients. AJR 177:85–89, Fig. 2)

the circumvallate papillae to the root of the epiglottis (see Fig. 4.2.2) (Grommet et al. 1982). Hypertrophy of the lingual tonsils may occur after puberty, as a compensatory response to tonsillectomy/adenoidectomy, or as non-specific response to allergy or repeated infection.

Hypertrophy of the lingual tonsils disrupts the normal reticular surface pattern. There are no radiographic criteria, however, to differentiate nodularity of the base of the tongue attributed to the normal lingual tonsil from that of lymphoid hyperplasia. On frontal views in patients with lymphoid hyperplasia, there are large 5-7 mm, smooth surfaced nodules carpeting the vertical surface of the tongue (Figs. 4.2.18 and 4.2.19a). In the lateral view, these nodules protrude posteriorly (Fig. 4.2.19b). With severe lymphoid hyperplasia, nodules may be detected in the valleculae, the lingual surface of the epiglottis and the upper hypopharynx. Although lymphoid hyperplasia can appear coarsely nodular, asymmetrically distributed or mass-like; if any asymmetric or mass-like nodularity is demonstrated radiographically, carcinoma or lymphoma should be excluded via ENT exam and/or MRI.



a **b**

Fig. 4.2.17a,b. Zenker's diverticulum and left lateral Killian-Jamieson diverticulum arising in same patient. **a** Spot radiograph obtained with patient in frontal position demonstrates a 3 cm barium-filled Zenker's diverticulum (*Z*) positioned slightly to the left of midline. Barium in right piriform sinus (*p*) results from reflux of barium from the Zenker's diverticulum back into the hypopharynx. A 1.6 cm left lateral, barium-filled Killian-Jamieson diverticulum (*K*) lies below and to the left of the Zenker's diverticulum. **b** Spot radiograph obtained with patient in steep right posterior oblique position shows that the Zenker's diverticulum (*Z*) extends posterior to the pharyngoesophageal segment (*arrowhead*). Part of the Killian-Jamieson diverticulum (*K*) lies anterior to the course of the proximal cervical esophagus (a and b reproduced with permission from Rubesin SE, Levine MS (2001) Killian-Jamieson diverticula: radiographic findings in16 patients. AJR 177:85–89 Fig. 3)

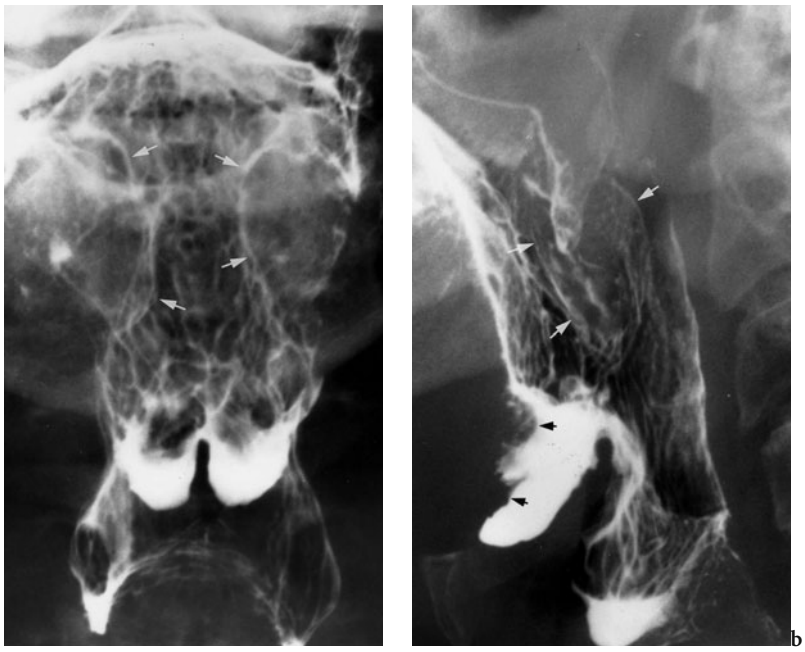
Fig. 4.2.18. Lymphoid hyperplasia, base of tongue. Frontal view shows many large, 5–7 mm nodules carpeting the surface of the base of the tongue (Reproduced with permission from Rubesin SE, Jessurun J, Robertson D (1987) et al. Lines of the pharynx. RadioGraphics 7:217–237, Fig. 9c)

4.2.4.2 Acute Pharyngitis

Acute epiglottitis usually affects children between 3 and 6 years of age, but may also be seen in adults (Harris et al. 1970). Plain film diagnosis is important, as manipulation of the tongue/pharynx or barium studies may exacerbate edema and trigger acute respiratory arrest (Balfe and Heiken 1986). Smooth enlargement of the epiglottis and aryepiglottic folds allows plain film diagnosis of severe epiglottitis.

Barium studies are usually not performed in immunocompetent patients with acute sore throat. In immunocompromised patients, barium studies are used to demonstrate the presence, type and severity of esophagitis. Thus, in patients with AIDS, a double-contrast examination may demonstrate the small ulcers of herpetic pharyngitis or the plaques of Candida pharyngitis (Fig. 4.2.20) (Rubesin and Glick 1988). Acute inflammatory disorders may cause laryngeal penetration due to abnormal pharyngeal elevation, epiglottic tilt or laryngeal closure.

Videopharyngography may be performed, however, on patients with acute odynophagia or dysphagia after trauma or suspected iatrogenic trauma. A



a **b**

Fig. 4.2.19a,b. Lymphoid hyperplasia of palatine tonsils and base of tongue. **a** Frontal view demonstrates large left and right palatine tonsils (*arrows*) protruding into oropharynx. The reticular surface of the base of the tongue is slightly prominent and the nodules are slightly enlarged. **b** Lateral view obtained while the patient sings "Eeee…" shows a slightly nodular mass (*white arrows*) in the tonsillar fossa. Several nodules (*black arrows*) protrude into the barium pooling in the valleculae (a and b reproduced with permission from Rubesin SE. Structural abnormalities. In Gore RM, Levine MS, Laufer I (1994) Textbook of Gastrointestinal Radiology. Philadelphia, WB Saunders, pp. 244–276, Fig. 17–12)

Fig. 4.2.20. Candida pharyngitis. Frontal view of the pharynx shows numerous small nodules etched in white by barium. The nodules carpet the pharynx (Reproduced with permission from Rubesin SE. Structural abnormalities of the pharynx. In: Gore RM, Levine MS (eds) 2000 Textbook of Gastrointestinal Radiology. Philadelphia, WB Saunders, pp. 227–255, Fig. 15–14.)

non-ionic water-soluble contrast agent is given first, followed by ionic, water-soluble contrast if no laryngeal penetration is seen (Fig. 4.2.21). When no perforation is demonstrated with a water-soluble contrast agent, this author prefers to give high-density barium, as this form of barium sticks to the mucosal surface and is easier to detect in the extraluminal soft tissues than thin barium.

4.2.4.3 Chronic Inflammatory Conditions

In patients with acute corrosive ingestion, water-soluble contrast studies may be utilized to exclude perforation of the pharynx, esophagus or stomach. Corrosive ingestion can result in amputation of the uvula and epiglottis and diffuse ulceration. With healing and scarring, epiglottic and pharyngeal wall deformity results in pharyngeal dysmotility (Fig. 4.2.22).

Aphthous stomatitis and oropharyngeal ulceration with subsequent scarring may be seen in Behçet's syndrome, bullous pemphigoid, epidermolysis bullosa, Reiter's syndrome, and Stevens-Johnson syndrome (Bosma et al. 1968; Kabakian and Dahmash 1978).

a

b

Fig. 4.2.21a,b. Acute perforation during endoscopy. **a** Scout radiograph shows a large amount of air (arrows) in the retropharyngeal space. The posterior wall of the pharynx (*open arrow*) is deviated anteriorly. **b** Spot radiograph obtained while the patient swallows water-soluble contrast agent shows a small spurt of contrast (*arrow*) just above the cricopharyngeus (*open arrow*). The contrast spreads in the retropharyngeal space

Fig. 4.2.22a,b. Sequelae of corrosive ingestion. Twenty years prior to this examination, the patient had ingested lye. **a** Frontal view demonstrates a low and easily seen epiglottis (*white arrows*). The right piriform sinus is contracted; the left piriform sinus has a radiolucent band representing a scar (*black arrow*) crossing its lower portion. **b** Lateral view shows that the truncated epiglottis (*arrow*) is posteriorly displaced. A small amount of barium coats the anterior wall of the laryngeal vestibule (a and b reproduced with permission from Rubesin SE (2000) Pharynx. In Levine MS, Rubesin SE, Laufer I (eds) Double contrast gastrointestinal radiology. Philadelphia, WB Saunders, pp. 60–89, Fig. 4–20)

Amputation of the uvula and tip of the epiglottis may be detected radiographically (Bosma et al. 1968).

4.2.4.4 Webs

Webs are thin folds of epithelium and lamina propria most frequently found on the anterior wall of the lower hypopharynx and proximal cervical esophagus (Clements et al. 1974). Pharyngeal and cervical esophageal webs are seen in 3-8% of patients undergoing an upper gastrointestinal examination and in up to 16% of patients at autopsy (Seaman 1967; Clements et al. 1974; Nosher et al. 1975; Ekberg 1981; Ekberg and Nylander 1983b). Some webs are the result of diseases that cause chronic inflammation and scarring, such as epidermolysis bullosa and benign mucous membrane pemphigoid. Webs in the valleculae or piriform sinuses may be normal variants (Ekberg et al. 1986). This author believes that some cervical esophageal webs are related to chronic gastroesophageal reflux, similar to the association of distal esophageal webs and gastroesophageal reflux disease (Weaver et al. 1984). The association of cervical esophageal webs, iron deficiency anemia, and pharyngeal or esophageal carcinoma – the Plummer-Vinson or Patterson-Kelly Syndrome – is controversial (Waldenstrom and Kjeulberg 1939; Mcnab-Jones 1961).

Webs are manifested radiographically as 1-2 mm in height, shelf-like radiolucent filling defects on the anterior wall of the pharyngoesophageal segment or proximal cervical esophagus. Larger webs may protrude deeply into the lumen or extend circumferentially around the wall of the cervical esophagus (Fig. 4.2.23). Dilatation of the pharynx or cervical esophagus proximal to the web or a stream of barium spurting through the web (the "jet phenomenon") are signs of partial obstruction and usually seen in symptomatic patients (Shauffer et al. 1977; Taylor et al. 1990). Webs should not be confused with redundant mucosa just posterior to the cricoid cartilage or a prominent cricopharyngeus (Fig. 4.2.24).

4.2.5 Tumors

4.2.5.1 Benign Tumors

A wide variety of benign tumors originate in the pharynx (Hyams et al. 1988; Bachman 1978). Retention cysts are lined by squamous epithelium and filled with desquamated debris. These cysts are the most common benign tumors of the base of the tongue (Fig. 4.2.25) and aryepiglottic folds (Fig. 4.2.26) (Bachman 1978).

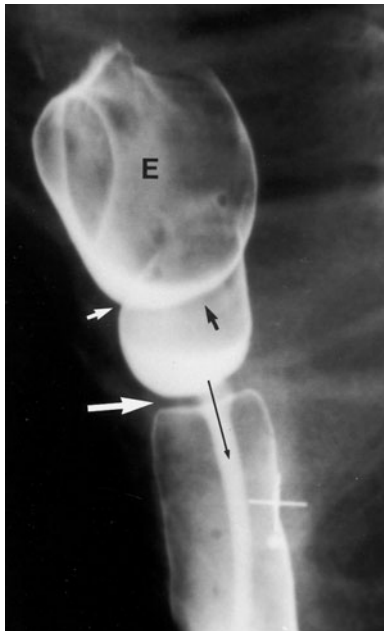


Fig. 4.2.23. Cervical esophageal webs. Spot radiograph obtained with the patient in the erect slightly right posterior oblique position shows two circumferential webs in the cervical esophagus. The upper web (*short white and black arrows*) is circumferential and only mildly compromises the lumen. The lower web (*large white arrow*) is also circumferential, but occludes more than two-thirds of the luminal diameter. Partial obstruction is manifested by dilatation of the proximal cervical esophagus (*E*) and the "jet phenomenon", barium spurting (*long black arrow*) through the web (Reproduced with permission from Rubesin SE (1995) Oral and pharyngeal dysphagia. Gastro Clin North Am 1995; 24:331–352, Fig. 1)

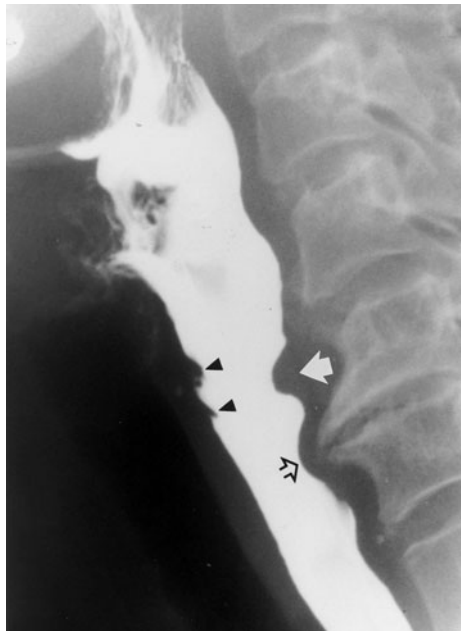


Fig. 4.2.24. Post-cricoid mucosa, prominent cricopharyngeus and cervical osteophyte impression. Spot radiograph obtained during drinking demonstrates osteophytes mildly impressing the upper cervical esophagus (*open black arrow*). Also note a mildly prominent cricopharyngeus (*white arrow*) and redundant mucosa posterior to the cricoid cartilage (*arrowheads*). None of these entities should be confused with cervical esophageal webs (Reproduced with permission from Rubesin SE (1995) Oral and pharyngeal dysphagia. Gastro Clin North Am 24:331–352, Fig. 7)

Ectopic thyroid tissue, thyroglossal duct cysts, granular cell tumors and benign tumors of minor mucoserous salivary gland origin also arise in the base of the tongue. Saccular cysts of the aryepiglottic folds are the mucus-filled variant of internal laryngoceles, arising from mucous secreting glands of the appendix of the laryngeal ventricle. Lipomas, neurofibromas, granular cell tumors and oncocytomas rarely also be seen in the aryepiglottic folds (Manson et al. 1978; Patterson et al. 1981; Hyams et al. 1988). Tumors related to neurofibromatosis (von Recklinghausen's disease) typically occur in the aryepiglottic folds (Fig. 4.2.27) and arytenoid cartilages (Chang-Lo 1977). Chondromas may originate in the posterior lamina of the cricoid cartilage (Hyams and Rabuzzi 1970).

Benign pharyngeal tumors are usually sessile submucosal masses, appearing radiographically en face as smooth, round, sharply circumscribed protrusions (see Fig. 4.2.26) and in profile as hemispheric lines with abrupt angulation to the luminal contour (see Fig. 4.2.25). (Balfe and Heiken 1986). Pedunculated tumors such as lipoma, papilloma or fibrovascular polyp are uncommon.

4.2.5.2 Squamous Cell Carcinoma

Squamous cell carcinomas comprise 90% of malignant pharyngeal lesions (Hyams et al. 1988; Cunningham and Catlin 1967). Multiple primary sites of squamous carcinoma are found in the oral cavity, pharynx, larynx, esophagus and lung in more than 20% of patients (Carpenter et al. 1976). Between 1 and 20% of patients with a head and neck squamous cell carcinoma will subsequently develop an esophageal squamous cell carcinoma (Goldstein and Zornoza 1978; Thompson et al. 1978)

Squamous cell carcinoma may be initially detected in patients undergoing pharyngoesophagography for pharyngeal symptoms or a palpable neck mass.

Fig. 4.2.25a,b. Retention cyst, left vallecula. **a** Spot radiograph obtained with patient in slight right posterior oblique position shows an ovoid, 1.5 cm radiolucent filling-defect (*arrows*) in the barium pool of the left vallecula. **b** Lateral view shows an ovoid area of increased radiodensity (*arrow*) etched by barium

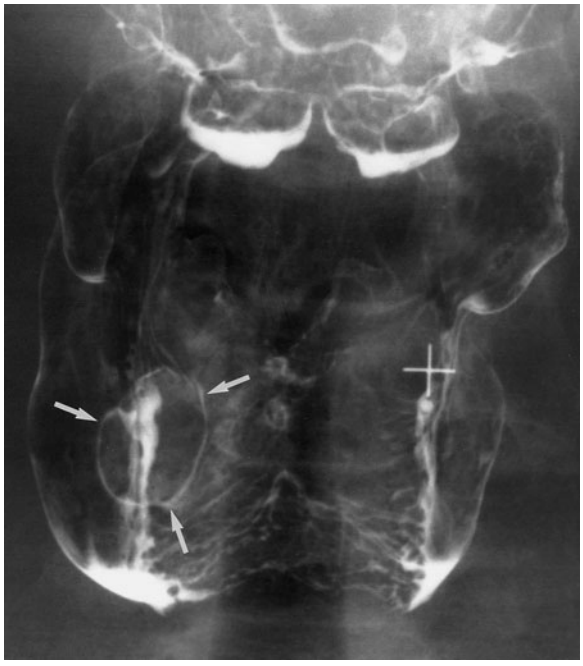
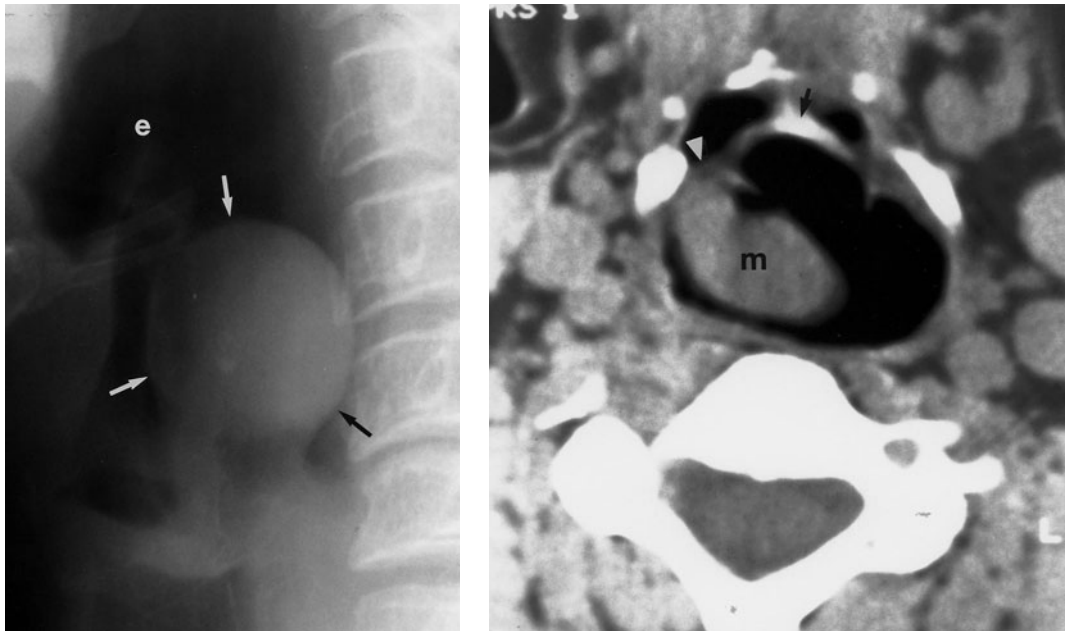


Fig. 4.2.26. Retention cyst arising in mucosa overlying the muscular process of the right arytenoid. A ring shadow (*arrows*) surrounding a 1.7 cm smooth-surfaced area of increased radiodensity is seen. This lesion had been missed on two previous endoscopies (Reproduced with permission from Rubesin SE, Glick SN (1988) The tailored double-contrast pharyngogram. Crit Rev Diagn Imaging 28:133–179 Fig 25a)

The symptoms are usually of short duration and include sore throat, dysphagia, odynophagia, choking or coughing. Hoarseness occurs in patients with laryngeal carcinoma, supraglottic cancers, or tumors infiltrating the arytenoid cartilage and medial pharyngeal wall. Most patients are 50 to 70 years of age and almost all abuse alcohol and tobacco.

In patients with known pharyngeal carcinoma, pharyngoesophagography assists in planning patient work-up and therapy. A pharyngogram establishes baseline motility and helps define the size, extent and inferior border of the tumor. Barium examination can exclude a synchronous esophageal tumor or a structural disorder (Zenker's diverticulum) that may change the way endoscopy is performed or the patient is treated. Pharyngography cannot be used to screen for pharyngeal cancer, as small, flat tumors can be missed in the region of the palatine tonsils or base of tongue, areas of normal mucosal nodularity. Below the pharyngoesophageal fold, however, barium studies detect 95% of structural abnormalities (Semenkovich et al. 1985). Barium studies are especially valuable in the deep valleculae, lower hypopharynx and pharyngoesophageal segment, areas that are difficult to visualize at endoscopy (Fig. 4.2.28).

The radiographic findings of pharyngeal cancer are similar to tumors elsewhere in the gastrointestinal



a b

Fig. 4.2.27a,b. Neurofibroma in 27-year-old-man with known neurofibromatosis and stridor. **a** Plain radiograph of the neck with patient in the lateral position shows a large, round soft-tissue mass (*arrows*) in the vicinity of the aryepiglottic folds. The epiglottic tip (*e*) is identified. **b** Axial image from CT scan shows a soft-tissue mass (*m*) arising from the right aryepiglottic fold (*arrowhead*). The tip of the epiglottis (*small arrow*) is calcified (A reproduced with permission from Rubesin SE, Laufer I (1991) Pictorial review: principles of double contrast pharyngography. Dysphagia 6:170–178, Fig. 1a; b reproduced with permission from Rubesin SE. Structural abnormalities. In Gore RM, Levine MS, Laufer I (1994) Textbook of Gastrointestinal Radiology. Philadelphia, WB Saunders, pp. 244–276, Fig. 17–17b)

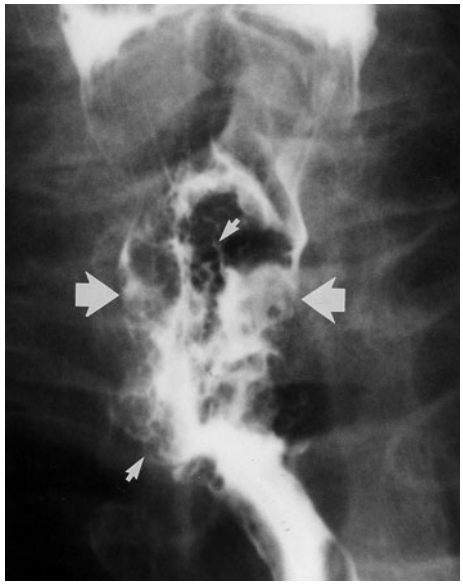


Fig. 4.2.28 Carcinoma of the pharyngoesophageal segment missed at ENT endoscopy. Spot radiograph shows mucosal nodularity (*representative areas of nodularity identified by small arrows*) involving the distal hypopharynx, pharyngoesophageal segment and upper cervical esophagus. The pharyngoesophageal segment is expanded (*large arrows*) by tumor. This patient had two ENT endoscopies within three months of the pharyngoesophagram

tract (Rubesin and Glick 1988). A mass protruding intraluminally may be manifested as loss of the normal expected contour, a focal area of increased radiodensity replacing the original air of the lumen, extra barium-coated lines protruding into the pharyngeal air column (Fig. 4.2.29), or a radiolucent filling defect in the barium pool (Fig. 4.2.30) (Jing 1970; Balfe and Heiken 1986; Rubesin 2000a; Rubesin and Laufer 1991). Mucosal ulceration is manifested as shallow pools of barium. Surface irregularity is manifested as a granular, finely nodular or lobulated surface texture (Fig. 4.2.31). Asymmetric motion may result from fixation of structures due to infiltrating tumor (Fig. 4.2.32). Asymmetric distensibility may result from an extrinsic mass impinging on the pharynx. In some patients with pharyngeal carcinoma, a focal area of mucosal nodularity is the only radiographic finding (Figs. 4.2.33 and Fig. 4.2.34).

The palatine tonsil is the most frequent site of pharyngeal squamous cell carcinoma. Exophytic tumors are easily seen on barium studies. Small infiltrative or ulcerative tumors may be missed in the normally nodular surface of the palatine tonsils. Tonsillar tumors spread to the soft palate, base of tongue and posterior pharyngeal wall. Cervical nodal metastases are found in one-half of patients (Balfe and Heiken 1986).

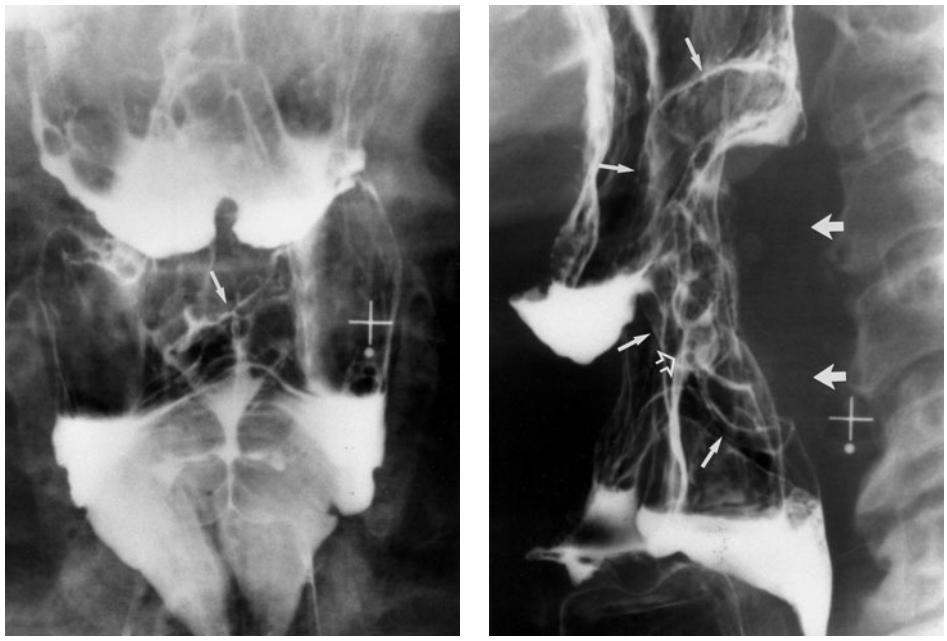


Fig. 4.2.29a,b. Radiographic findings of pharyngeal cancer and the value of obtaining multiple projections. **a** Frontal view of the pharynx shows a few barium-etched nodules in the upper hypopharynx (*arrow*). Aspirated barium coats the false and true vocal cords. There is moderate stasis in the hypopharynx. **b** Lateral view demonstrates a large mass arising from the posterior pharyngeal wall as the explanation for the subtle mucosal nodularity seen on the frontal view. The mass if manifested as loss of the normal contour and soft tissue density partially replacing the air shadow of the lower oropharynx and upper hypopharynx (*large arrows*), barium etched lines within the lumen (*thin arrows*) and focal mucosal nodularity (*open arrow*). Barium coating the laryngeal vestibule and upper trachea and stasis in the lower hypopharynx is the result of pharyngeal dysmotility (a and b reproduced with permission from Rubesin SE, Glick SN (1988) The tailored double-contrast pharyngogram. Crit Rev Diagn Imaging 28:133–179, Fig. 30a and 30b)

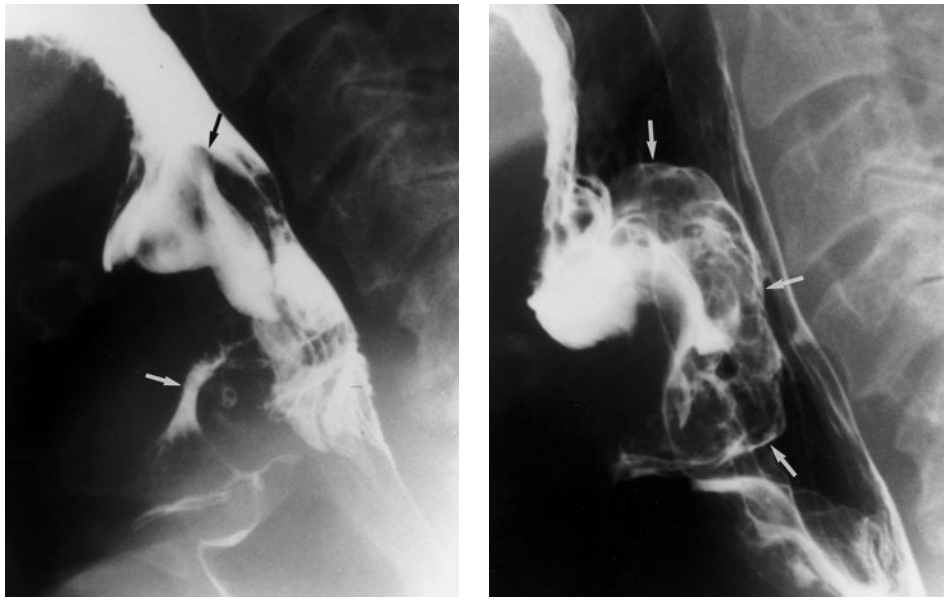


Fig. 4.2.30a,b. a Spot radiographs vs. dynamic imaging in patient with large supraglottic carcinoma. Image obtained during dynamic imaging shows lack of epiglottic tilt (*black arrow*) and probable epiglottic enlargement. Barium enters the laryngeal vestibule (*white arrow*) during swallowing, with subsequent coating of the laryngeal ventricle and trachea. **b** Spot radiograph demonstrates massive enlargement of the epiglottis (*arrows*). The epiglottic mucosa is markedly nodular. These images show how spot radiographs demonstrate morphology better than dynamic images, but dynamic images demonstrate motility (Reproduced with permission from Rubesin SE (1991) Pharyngeal dysfunction. In Gore RM (ed) Categorical course on gastrointestinal radiology. Reston, VA, American College of Radiology, pp. 1–9. Fig. 11c and 11d)

Fig. 4.2.31a,b. Mucosal nodularity as a sign of cancer. **a** Frontal view shows diffuse narrowing of the lumen (*arrows*) of the lower hypopharynx, pharyngoesophageal segment and upper cervical esophagus. The normal tips of the piriform sinuses are obliterated. **b** Lateral view obtained during patient drinking shows numerous radiolucent nodules (*representative nodules identified by arrows*) in the barium bolus. The tumor involves base of tongue, the anterior wall of the laryngeal vestibule, the entire glottis, the anterior wall of the lower hypopharynx

Squamous cell cancers of the base of the tongue often present as deeply infiltrative, advanced lesions with nodal metastases (Frazell and Lucas 1962; Strong 1979). Lymph node metastases are detected ipsilaterally or bilaterally in more than 70% of patients. Exophytic lesions protrude into the oropharyngeal airspace (Jiminez 1970; Apter et al. 1984). Ulcerated lesions appear as irregular barium collections extending deep to the expected contour of the base of the tongue. Tumor spreads to the palatine tonsil, valleculae, or pharyngoepiglottic fold. Small plaque-like or ulcerative lesions may be hidden in the valleculae or in the recess between the tongue and tonsil.

Supraglottic carcinomas are tumors that arise in the epiglottis (see Fig. 4.2.30), aryepiglottic folds, mucosa overlying the muscular processes of the arytenoid cartilages (see Fig. 4.2.32), false vocal cords and laryngeal ventricles. Supraglottic carcinomas spread rapidly through the rich lymphatic network of the supraglottic space. Ulcerative lesions penetrate the tongue, valleculae, and pre-epiglottic space, pharyngoepiglottic folds and lateral pharyngeal walls (Seaman 1974). A paucity of lymphatics near the true vocal cords limits the spread of supraglottic tumors through the laryngeal ventricles into the true vocal folds. Cervical lymph node metastases are found in 30-50% of patients (Balfe and Heiken 1986; Kirchner and Owen 1977).

Piriform sinus carcinomas are bulky, exophytic lesions that spread quickly and metastasize widely. Medial wall tumors spread to the aryepiglottic fold, arytenoid and cricoid cartilages, and the paraglottic space (Jing 1970; Johnson 1995). Lateral wall tumors (see Fig. 4.2.33) may invade the thyrohyoid membrane, thyroid cartilage, and soft tissues of the neck, including the carotid sheath (Zbaren and Egger 1997). Lymph node metastases are found in 70-80% of patients (Balfe and Heiken 1986; Silver 1972).

Carcinomas of the posterior pharyngeal wall are typically long fungating lesions that may spread vertically into the nasopharynx or cervical esophagus. Jugular or retropharyngeal lymphatic metastases are found in 50% of patients (Carpenter et al. 1976).

Post cricoid squamous cell carcinomas are uncommon, annular, infiltrating lesions that may extend into

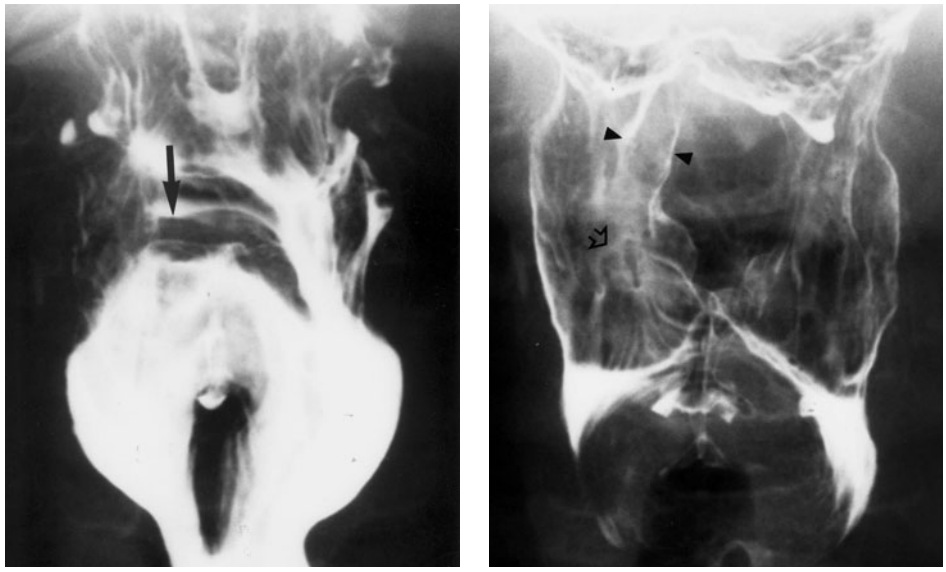


Fig. 4.2.32a–c. a Value of spot radiographs. Frontal view obtained at the end of bolus passage through the pharynx shows diminished epiglottic tilt on the right (*arrow*). **b, c.** Spot radiographs obtained in frontal (**b**) and slight right posterior oblique (**c**) position shows moderate thickening of the right aryepiglottic fold (*arrowheads*), mild enlargement and tumor nodularity involving the mucosa overlying the muscular process of the right arytenoid cartilage (*open arrow*) (a, b, c reproduced with permission from Rubesin SE, Pharyngeal dysfunction. In Gore RM (ed) (1991) Categorical course on gastrointestinal radiology. Reston, VA, American College of Radiology, 1–9, Fig. 12a, 12b, 12c)

the hypopharynx or cervical esophagus (see Fig. 4.2.28). In Scandinavia, these are the tumors that have been associated with Plummer-Vinson syndrome.

4.2.5.3 Lymphoma

Lymphomas comprise about 10% of pharyngeal malignancies (Banfi et al. 1970). Almost all pharyngeal lymphomas are non-Hodgkin's lymphoma, arising in the palatine or lingual tonsils or the adenoids. Pharyngeal involvement in Hodgkin's disease occurs in only 1-2% of patients, despite the fact that Hodgkin's disease often begins in cervical lymph nodes (Todd and Michaels 1974).

Lymphoma of the pharynx occurs in the palatine tonsils in 40-60% of patients, the nasopharynx in 18-28%, and the base of the tongue in 10% (Hyams et al. 1988; Al-Saleem et al. 1970). Multiple sites of involvement are seen in 25% of patients. Fifteen percent of patients have bilateral palatine tonsillar involvement (Banfi et al. 1970). Lymphomas only rarely arise in the hypopharynx. At the time of initial diagnosis, the cervical lymph nodes are involved in 60% of patients, and 10% of patients have extranodal involvement.

Pharyngeal lymphomas typically appear as large, lobulated masses involving the nasopharynx, palatine tonsil (Fig. 4.2.35), base of tongue, or a combination of these locations (Fig. 4.2.36) (Hyams et

Fig. 4.2.33a,b. Early squamous cell carcinoma manifested as tumor nodularity. **a** Frontal view shows subtle, focal nodularity (*arrows*) in the lower right piriform sinus. **b** Lateral view demonstrates a focal are of nodularity along the anterior wall of the hypopharynx (*arrows*). This 64-year-old woman had a tip of the tongue squamous cell carcinoma and was being studied to exclude an esophageal carcinoma prior to surgery (a and b reproduced with permission from Levine MS, Rubesin SE (1990) Update on esophageal radiology. AJR 155:933–941, Fig. 4)

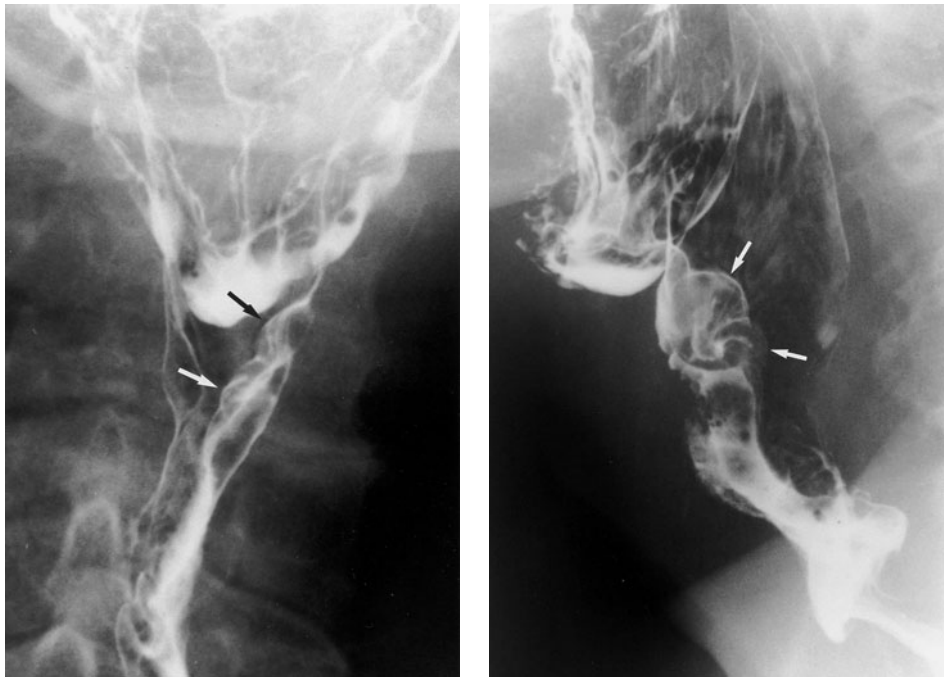


Fig. 4.2.34. Recurrent squamous cell carcinoma two years after total laryngectomy and radiation therapy. **a** Frontal view shows large nodules (*arrows*) altering the normally smooth contour of the neopharyngeal tube. **b** Lateral view shows a mass (*arrows*) etched-in-white by barium just below the level of the neovalleculae. The posterior neopharyngeal wall is poorly coated by barium

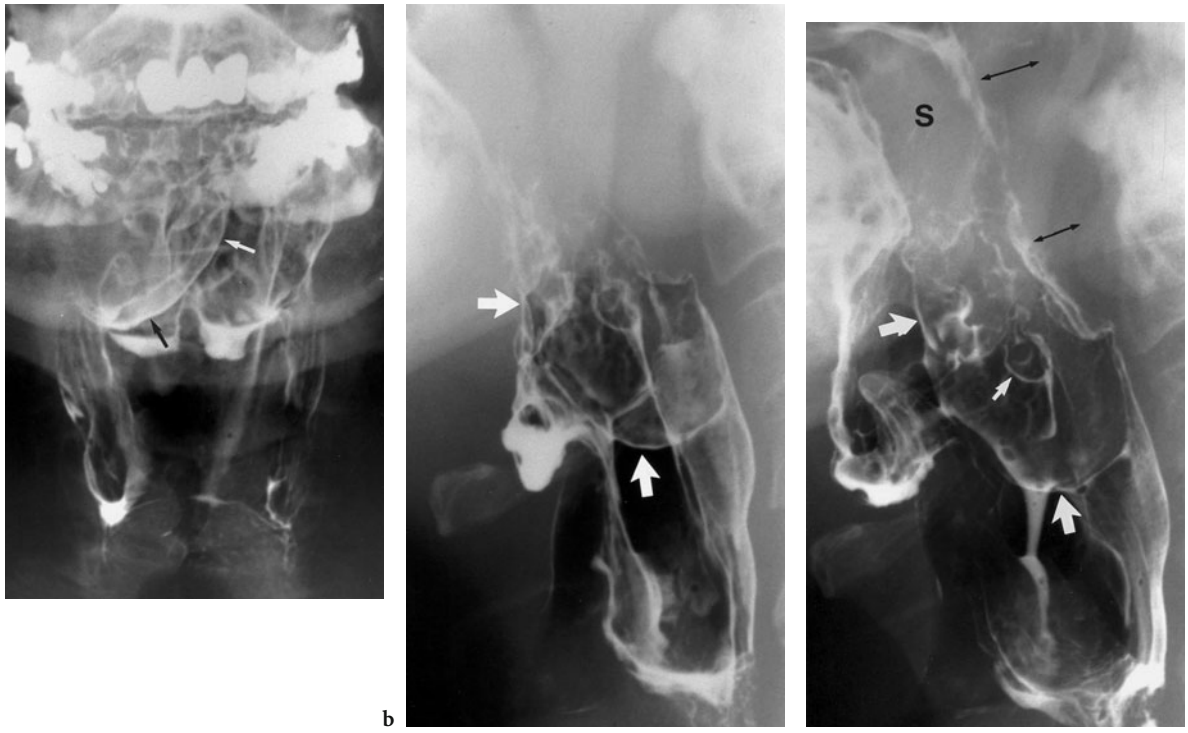


Fig. 4.2.35a–c. Lymphoma of pharynx and the value of phonation. **a** Frontal view shows a soft tissue mass (*arrows*) obliterating the right oropharyngeal wall. **b** Lateral view shows a soft tissue mass (*arrows*) in the oropharynx. **c** Lateral view obtained after instillation of 1 ml barium into each naris, and having the patient phonate Eeee . . . The mass (*large arrows*) has a smooth surface inferiorly and a central ring (*small arrow*) demarcating an empty ulcer. The mass is clearly separated from the base of the tongue by the patient's phonation. Tumor extension into the soft palate and posterior pharyngeal wall is manifested as enlargement and nodularity of the soft palate (*s*) and enlargement of the retropharyngeal space and nodularity of its surface (*double arrows*) (Reproduced with permission from Levine MS, Rubesin SE (1990) Radiologic investigation of dysphagia. AJR 154:1152–1163)

Fig. 4.2.36a,b Lymphoma of tongue and right palatine tonsil. **a** Frontal view shows a mass (*arrows*) manifested by increased soft tissue density and tumor nodularity. **b** Right posterior oblique view shows tumor nodularity en face involving the right palatine fossa (*small arrows*) and base of the tongue (*large arrow*)

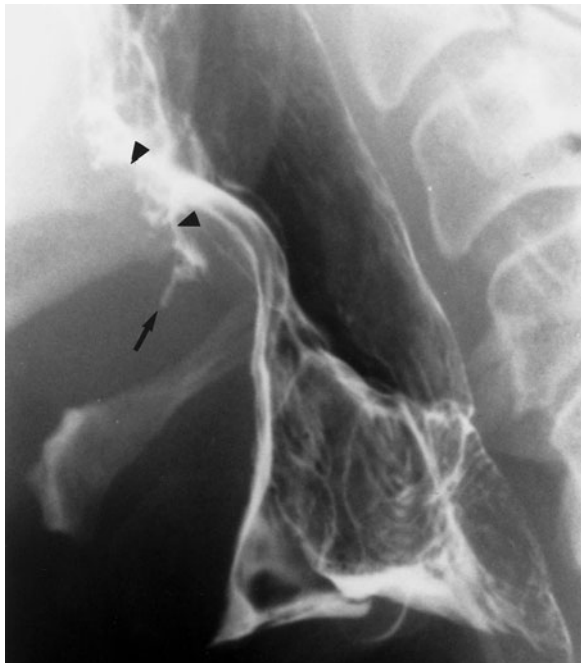


Fig. 4.2.37. Adenocarcinoma of base of tongue. Lateral view shows obliteration of the valleculae (*arrow*) and nodularity of the base of tongue (*arrowheads*). Barium coating the laryngeal vestibule and ventricle resulted from absent epiglottic tilt. The tumor presumptively arose in submucosal glands in the base of the tongue (Reproduced with permission from Rubesin SE. Structural abnormalities. In Gore RM, Levine MS, Laufer I (1994) Textbook of Gastrointestinal Radiology. Philadelphia, WB Saunders, pp. 244–276, Fig. 17–37)

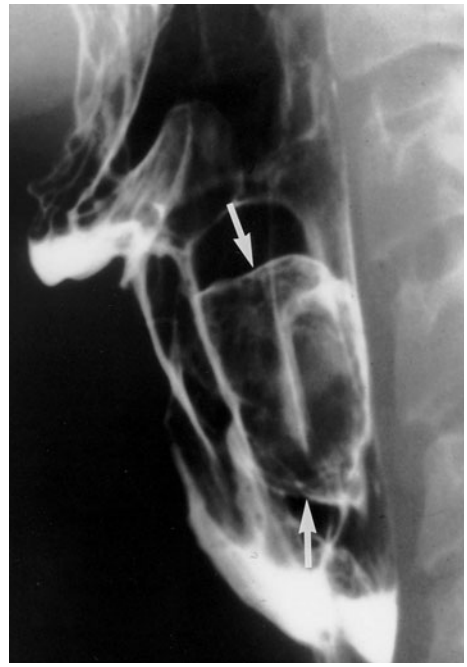


Fig. 4.2.38. Kaposi sarcoma. Lateral view shows a relatively smooth-surfaced mass (*arrows*) in the air space of the upper hypopharynx (Courtesy of Dean D.T. Maglinte, M.D., University of Indiana)

al. 1988; Rubesin 2000c). The bulging submucosal masses obliterate normal lymphoid follicular pattern of the base of the tongue or palatine tonsil (Rubesin 2000c).

4.2.5.4 Other Malignant Tumors

The minor mucoserous salivary glands scattered deep to the epithelial layer of the pharynx give rise to tumors of diverse histology and clinical course. Most (65-88%) minor salivary gland tumors are malignant (Spiro et al. 1973; Conley and Dingman 1974). The most frequent malignant minor salivary tumors are adenoid cystic carcinoma (35%), solid adenocarcinoma (22%) (Fig. 4.2.37), and mucoepidermoid carcinoma (16%) (Spiro et al. 1973). Most pharyngeal minor salivary tumors arise in the soft palate. Palatal salivary gland tumors spread directly to the tongue, submandibular gland, mandible, and lingual and hypoglossal nerves. Adenoid cystic carcinoma typically spreads via a perineural route. Cervical metastases are infrequent, occurring in 23% of patients with malignant minor salivary tumors.

Synovial sarcomas of the pharynx are extremely rare (Krugman et al. 1973). These lesions are large, bulky tumors involving the larynx, pharynx and soft tissues of the neck (Gatti et al. 1975).

Kaposi sarcoma may detected in the pharynx in patients with AIDS. Kaposi sarcoma has a wide range of radiographic appearances, including small nodules, plaque-like lesions, small submucosal masses with or without central ulceration or larger polypoid masses (Fig. 4.2.38) (Emery et al. 1986).

The pharynx may be invaded by a variety of tumors arising in the laryngeal cartilages, most frequently the cricoid cartilage. These cartilaginous tumors include chondroma, osteochondroma and chondrosarcoma (Huizenga and Balogh 1970). Radiographically, the lower hypopharynx and pharyngoesophageal segment is compressed by a smooth-surfaced mass arising in the posterior lamina of the cricoid cartilage. In more than 80% of patients, stippled calcification is detected centrally or peripherally (Hyams et al. 1988).

References:

Al-Saleem T, Harwick R, Robbins R, et al. (1970) Malignant lymphomas of the pharynx. Cancer 26:1769–1778

Apter AJ, Levine MS, Glick SN (1984) Carcinomas of the base of the tongue: diagnosis using double-contrast radiography of the pharynx. Radiology 151:123–126

Bachman AL (1978) Benign non-neoplastic conditions of the larynx and pharynx. Radiol Clin North Am 16:273–290

Bachman AL, Seaman WB, Macken KL (1968) Lateral pharyngeal diverticula. Radiology 91:774–782

Balfe DM, Heiken JP (1986) Contrast evaluation of structural lesions of the pharynx. Curr Probl Diagn Radiol 15: 73–160

Banfi A, Bonadonna G, Carnevali G, et al. (1970) Lymphoreticular sarcomas with primary involvement of Waldeyer's ring. Cancer 26:341–351

Bosma JF, Gravkowski EA, Tyrostad CW (1968) Chronic ulcerative pharyngitis. Arch Otolaryngol 87:85–96

Brady AP, Stevenson GW, Somers S (1995) Premature contraction of the cricopharyngeus: new sign of gastroesophageal reflux disease. Abdom Imaging 20:225–229

Carpenter RJ III, DeSanto LW, Devine KD, et al. (1976) Cancer of the hypopharynx. Arch Otolaryngol 102:716–721

Chang-Lo M (1977) Laryngeal involvement in Von Recklinghausen's disease. Laryngoscope 87:435–442

Clements JL, Cox GW, Torres WE, et al. (1974) Cervical esophageal webs–a roentgen-anatomic correlation. AJR 121:221–231

Conley J, Dingman DL (1974) Adenoid cystic carcinoma in the head and neck (cylindroma). Arch Otolaryngol 100:81–90

Cunningham MP, Catlin D (1967) Cancer of the pharyngeal wall. Cancer 20:1859–1866

Curtis DJ, Cruess DF, Crain M, et al. (1988) Lateral pharyngeal outpouchings: a comparison of dysphagic and asymptomatic patients. Dysphagia 2:156–161

Delahunty JE, Margulies SE, Alonso UA, et al. (1971) The relationship of reflux esophageal to pharyngeal pouch (Zenker's diverticulum). Laryngoscope 81:570–577

DuBrul EL (1980) Sicher's oral anatomy, 7th edn. CV Mosby, St. Louis, pp 319–350

Ekberg O (1981) Cervical esophageal webs in patients with dysphagia. Clin Radiol 32:633–641

Ekberg O, Nylander G (1983a) Lateral diverticula from the pharyngoesophageal junction area. Radiology 146: 117–122

Ekberg O, Nylander G (1983b) Webs and web-like formations in the pharynx and cervical esophagus. Diagn Imaging 52: 10–18

Ekberg O, Birch-lensen M, Lindstrom C (1986) Mucosal folds in the valleculae. Dysphagia 1:68–72

Emery CD, Wall S, Federle MP, et al. (1986) Pharyngeal Kaposi's sarcoma in patients with AIDS. AJR 147:919–922

Frazell EL, Lucas JC (1962) Cancer of the tongue: report of the management of 1554 patients. Cancer 15:1085–1099

Frieling T, Berges W, Lubke HJ, et al. (1988) Upper esophageal sphincter function in patients with Zenker's diverticulum. Dysphagia 3:90–92

Gatti WM, Strom CG, Orfei E (1975) Synovial sarcoma of the laryngopharynx. Arch Otolaryngol 98:53–54

Goldstein HM, Zornoza J (1978) Association of squamous cell carcinoma of the head and neck with cancer of the esophagus. AJR 131:791–794

Gromet M, Homer MJ, Carter BL (1982) Lymphoid hyperplasia at the base of the tongue. Radiology 144:825–828

Harris RD, Berdon WE, Baker DH (1970) Roentgen diagnosis of acute epiglottis in the adult. J Can Assoc Radiol 21: 270–272

Huizenga C, Balogh K (1970) Cartilaginous tumors of the larynx. Cancer 26:201–210

Hyams VJ, Rabuzzi DD (1970) Cartilaginous tumors of the larynx. Laryngoscope 80:755–767

Hyams VJ, Batsakis JG, Michaels L (1988) Tumors of the upper respiratory tract and ear. In: Atlas of tumor pathology, Second series, Fascicle 25. Armed Forces Institute of Pathology, Bethesda, MD.

Jimenez JR (1970) Roentgen examination of the oropharynx and oral cavity. Rad Clin North Am 8:413–424

Jing BS (1970) Roentgen examination of the larynx and hypopharynx. Radiol Clin North Am 8:361–386

Johnson JT, Bacon GW, Meyers EN, Wagner RL (1995) Medial vs. lateral wall piriform sinus carcinomas: implications for management of regional lymphatics. Head Neck 16:401–405

Kabakian HA, Dahmash MS (1978) Pharyngoesophageal manifestations of epidermolysis bullosa. Clin Radiol 29: 91–94

Killian G (1908) Ueber den Mund der Speiseröhre. Zeitschr Ohrenheilkd Wiesb 55:1–41

Kirchner JA, Owen JR (1977) Five hundred cancers of the larynx and piriform sinuses: results of treatment by radiation and surgery. Laryngoscope 87:1288–1303

Knuff TE, Benjamin SB, Castell DO (1982) Pharyngoesophageal (Zenker's) diverticulum: a reappraisal. Gastroenterology 82:734–736

Krugman ME, Rosin HD, Toker C (1973) Synovial sarcoma of the laryngopharynx. Arch Otolaryngol 98:53–54

Levine MS, Rubesin SE (1990a) Update on esophageal radiology. AJR 155:933–94

Levine MS, Rubesin SE (1990b) Radiologic investigation of dysphagia. AJR 154:1157–1163

Lindbichler F, Raith J Uggowitzer M, Hausegger K. (1998) Aspiration resulting from lateral hypopharyngeal pouches. AJR 170:129–132

Mannsson T, Wilske J, Kindblom L-G (1978) Lipoma of the hypopharynx: a case report and a review of the literature. J Laryngol Otol 92:1037–1043

Maran AGD, Buchanan DR (1978) Branchial cysts, sinuses and fistulae. Clin Otolaryngol 3:407–414

McNab-Jones RF (1961) The Patterson-Brown-Kelly syndrome: its relationship to iron deficiency and postcricoid carcinoma. J Laryngol Otol 71:529–561

Nanson EM (1976) Carcinoma in a long-standing pharyngeal diverticulum. Br J Surg 63:417–419

Nosher JL, Campbell WL, Seaman WB (1975) The clinical significance of cervical esophageal and hypopharyngeal webs. Radiology 117:45–47

Patterson HC, Dickerson GR, Pilch BZ, et al. (1981) Hamartoma of the hypopharynx. Arch Otolaryngol 107:767–772

Pernkopf E (1989) Anatomy, vol 1, Head Neck, 3rd edn. Urban & Schwarzenberg, Baltimore

Perrot JW (1962) Anatomical aspects of hypopharyngeal diverticula. Aust N Z J Surg 31:307–317

Pitman RG, Fraser GM (1965) The post-cricoid impression of the esophagus. Clin Radiol 16:34–39

Rubesin SE (1991) Pharyngeal dysfunction. In: Gore RM (ed) Categorical course on gastrointestinal radiology. American College of Radiology, Reston, VA, pp. 1–9.
Rubesin SE (1994) Structural abnormalities. In: Gore RM, Levine MS, Laufer I. Textbook of Gastrointestinal Radiology. WB Saunders, Philadelphia, pp. 244–276
Rubesin SE (1995) Oral and pharyngeal dysphagia. Gastro Clin North Am 24:331–352
Rubesin SE (2000a) Pharynx. In: Levine MS, Rubesin SE, Laufer I (eds) Double contrast gastrointestinal radiology. WB Saunders, Philadelphia, pp 61–89
Rubesin SE (2000b) Pharynx: normal anatomy and examination techniques. In: Gore RM, Levine MS (eds) Textbook of gastrointestinal radiology. WB Saunders, Philadelphia, pp 190–211
Rubesin SE (2000c) Structural abnormalities of the pharynx. In Gore RM, Levine MS (eds) Textbook of gastrointestinal radiology. WB Saunders, Philadelphia, pp 227–255
Rubesin SE, Glick SN (1988) The tailored double-contrast pharyngogram. Crit Rev Diagn Imaging 28:133–179
Rubesin SE, Laufer I (1991) Pictorial review: principles of double contrast pharyngography. Dysphagia 6:170–178
Rubesin SE, Stiles TD (1997) Principles of performing a «modified barium swallow» examination. In: Balfe DM, Levine MS (eds) Categorical course in diagnostic radiology: gastrointestinal. RSNA Publications, Oak Brook, IL, pp 7–20
Rubesin SE, Levine MS (2001) Killian-Jamieson diverticula: radiographic findings in 16 patients. AJR 177:85–89
Rubesin SE, Jessurun J, Robertson D, et al. (1987a) Lines of the pharynx. Radiographics 7:217–237
Rubesin SE, Jones BJ, Donner MW (1987b) Contrast pharyngography: the importance of phonation. AJR 148:269–272
Seaman WB (1967) The significance of webs in the hypopharynx and upper esophagus. Radiology 89:32–38
Seaman WB (1974) Contrast radiography in neoplastic disease of the larynx and pharynx. Semin Roentgenol 9:301–209
Semenkovich JW, Balfe DM, Weyman PJ, et al. (1985) Barium pharyngography: comparison of single and double contrast technique. AJR 144:715–720
Shauffer IA, Phillips HE, Sequeira J (1977) The jet phenomenon: a manifestation of esophageal web. AJR 129:747–748
Shirazi KK, Daffner RH, Gaede JT (1977) Ulcer occurring in Zenker's diverticulum. Gastrointest Radiol 2:117–118
Silver CE (1977) Surgical management of neoplasms of the larynx, hypopharynx and cervical esophagus. Curr Probl Surg 14:2–69
Smiley TB, Caves PK, Porter DC (1970) Relationship between posterior pharyngeal pouch and hiatus hernia. Thorax 25: 725–731
Spiro RH, Koss LG, Hajdu SI, et al. (1973) Tumors of minor salivary origin. Cancer 31:117–129
Strong EW (1979) Carcinoma of the tongue. Otolaryngol Clin North Am 12:107–114
Taylor AJ, Stewart ET, Dodds WJ (1990) The esophageal jet phenomenon revisited. AJR 155:289–290
Thompson WM, Oddson TA, Kelvin F, et al. (1978) Synchronous and metachronous squamous cell carcinoma of the head, neck and esophagus. Gastrointest Radiol 3:123–127
Todd GB, Michaels L (1974) Hodgkin's disease involving Waldeyer's lymph ring. Cancer 34:176–1768
Waldenstrom J, Kjellberg SR (1939) The roentgenological diagnosis of sideropenic dysphagia (Plummer-Vinson's syndrome) Acta Radiol 20:618–638
Weaver JW, Kaude JV, Hamlin DJ (1984) Webs of the lower esophagus: a complication of gastroesophageal reflux? AJR 142:289–292
Wychulis AR, Gunnulaugsson GH, Clagett OT (1969) Carcinoma arising in pharyngoesophageal diverticulum. Surgery 66:976–979
Zaino C, Jacobson HG, Lepow H, et al. (1967) The pharyngoesophageal sphincter. Radiology 89:639–645
Zaino C, Jacobson HG, Lepow H, et al. (1970) The pharyngoesophageal sphincter. Charles C. Thomas, Springfield, IL
Zbaren P, Egger C (1997) Growth patterns of piriform sinus carcinomas. Laryngoscope 107:511–518

4.3 Pharyngeal Manometry

Rolf Olsson

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4.3.1 Introduction

Dysphagia is the subjective awareness of swallowing difficulties. A normal swallow is the smoothly coordinated transportation of a bolus from the oral cavity, through the pharynx and esophagus, into the stomach. When normal it is the combination of voluntary, automatic and autonomic functions (Miller 1986). Abnormalities in the oral cavity, pharynx and esophagus may hamper normal swallow, as may impairment of the neurologic control (Bosma et al. 1986; Buchholz et al. 1985; Gordon et al. 1987). Swallowing complaints are common, especially in hospitalized patients. Several studies have reported up to 30% of these populations to suffer from dysphagia (Groher and Bukatman 1986; Lindgren and Janzon 1991).

In the examination of the dysphagic patient several diagnostic modalities are available. The clinical and neurological examination is important, followed by radiographic and/or endoscopic examination for assessment of morphological changes (Dodds et al. 1990; Ekberg and Wahlgren 1985). When this is ruled out, more functional investigations come into place, such as dynamic barium swallow, pH monitoring, manometry, scintigraphy, ultrasound and electromyography.

R. Olsson, MD, PhD
Department of Diagnostic Radiology, Malmö University Hospital, SE-205 02 Malmö, Sweden

Dynamic cine- and videoradiography of pharyngeal barium swallow provides morphological as well as qualitative functional information of the swallowing sequence (Ekberg 1987; Ekberg and Nylander 1982). However, dynamic barium swallow relies mainly on functional qualitative judgment, failing to quantify the results. This is for example the case when trying to assess pharyngeal paresis in terms of degree of barium retention in the pharynx.

In this context, pharyngeal manometry is capable of providing a more quantitative analysis of pharyngeal muscle function in terms of intraluminal pressure registration. Concurrent barium swallow and pharyngeal manometry combines the qualitative assessment of bolus transport with quantitative registration of the contractions of the pharyngeal wall.

The previously described results with combined manometry and videoradiography in the literature are promising. Recent advances in manometric technology together with computer analysis of the data have facilitated pharyngeal manometry and the combined technique has been advocated by several authors.

4.3.2 Manometry

Pressure is the force acting perpendicular to an area. Physiological pressures are usually expressed in millimetres of mercury (mm Hg). Many other pressure units are available, for example Pascal (Pa), Newton/m^2, cm of H_20. Manometry records intraluminal pressure activity and detects and quantifies changes in intraluminal pressures that in the pharynx are caused by contractions of the muscles of the gullet. Such intraluminal pressures can be described as either contact pressures, when the manometric sensor is in direct contact with the pharyngeal wall, or cavity pressures when the sensor is completely surrounded by air or fluid. The latter is also called intrabolus pressure (Brasseur 1987; Brasseur and Dodds 1991).

Intraluminal esophageal manometry has been used extensively as a test of esophageal function for more than 100 years (Kronecker and Meltzer 1883). In the early history of manometry, pressures were obtained by means of static water-filled catheters within the esophagus. Around 20 years ago the development of low-compliance perfusion systems contributed to more accurate measurements of intraluminal pressure and motility (Arndorfer et al. 1977). The continuous perfusion prevented occlusion of the sensors.

Solid-state intraluminal transducers have developed during the last decade and have further improved the manometric accuracy (Castell and Castell 1993). These sensors are located on the catheter and can be placed in the gullet with direct measurement on the site of interest.

Together with the advances in computer technology, analysis and storage of manometric data have been facilitated.

4.3.3. Pharyngeal Manometry

Pharyngeal manometry is a less frequently used technique with difficulties in pressure recording because of the rapid pharyngeal swallowing sequence, as well as the movement of the soft palate and the larynx and also the radial asymmetry of intraluminal contact pressure (Welch et al. 1979; Winans 1972). Perfused manometry is not suitable in the pharynx because of the continuous flow that will trigger swallows.

Furthermore, perfused manometry has to be performed in a supine position because of the need to calibrate the sensors at the same level. Pharyngeal manometry in an upright physiologic position, with accurate recording technique, makes solid-state manometry a prerequisite in pharyngeal manometry (Orlowski et al. 1982).

Manometric evaluation of the pharynx with solid-state manometry and computerized analysis has been described in several papers from Don Castell and his group in Philadelphia (Castell and Castell 1993; Castell et al. 1993; Castell et al. 1990). They have thoroughly described the difficulties in pharyngeal manometry due to the rapid contractions of the striated muscles in the pharynx and the radial asymmetry with higher pressure in the antero-posterior direction. This group has also reported normal values in pharyngeal manometry in terms of contraction and relaxation but also in timing and coordination between events during pharyngeal swallow.

We have established normal values in pharyngeal manometry with simultaneous videoradiography and fluoroscopic control of sensor positioning (Olsson et al. 1995a) (Figs. 4.3.1 and 4.3.2). We included 25 non-dysphagic volunteers with normal videoradiographic parameters in this study. The examination was performed in an upright physiologic position during 10-ml barium and dry swallows. Mean resting pressure in the upper esophageal sphincter was 90±33 (±2 SD) mm Hg. Mean residual pressure during relaxation of the upper esophageal sphincter was 7.2±8.0 (±2 SD) mm Hg during barium swallow and 3.8±6.2 (±2 SD) mm Hg during dry swallow. The mean duration of upper esophageal sphincter relaxation was 601±248 (±2 SD) ms.

The mean peristaltic contraction of the upper esophageal sphincter was 254±143 (±2 SD) mm Hg. Fourteen (56 %) of the 25 had a measurable intrabolus pressure (mean 33±17 mm Hg) at the level of the inferior pharyngeal constrictor.

A specific finding was discovered when the epiglottis tilts down hitting the manometric sensor. This epiglottic tilt was identified in seven subjects (28%) and caused pressures around 600 mm Hg. A standardized manometric technique is important in videomanometry and normal values as described in this study are essential in clinical use.

4.3.4. Technical Considerations in Pharyngeal Manometry

Pharyngeal pressures are usually expressed in millimetres of mercury (Hg) and they are normally expressed relative to atmospheric pressure. Changes in atmospheric pressure seldom have an effect on physiological pressures but calibration and establishing a “zero” pressure is necessary to achieve reproducible and comparative data.

In 1982 Orlowski described the requirements for accurate recording of pharyngeal pressures. He studied the frequency characteristics of pharyngeal pressure complexes in normal subjects and found a recording accuracy of up to 48 Hz needed for high fidelity recorded in the pharynx. Frequency content of upper esophageal pressure has also been reported by Ask (1979).

A low compliance perfusion system has a bandwidth of approximately only 23 Hz (Ask et al. 1979). Orlowski et al. (1982) concluded that solid-state sensors are needed in pharyngeal manometry.

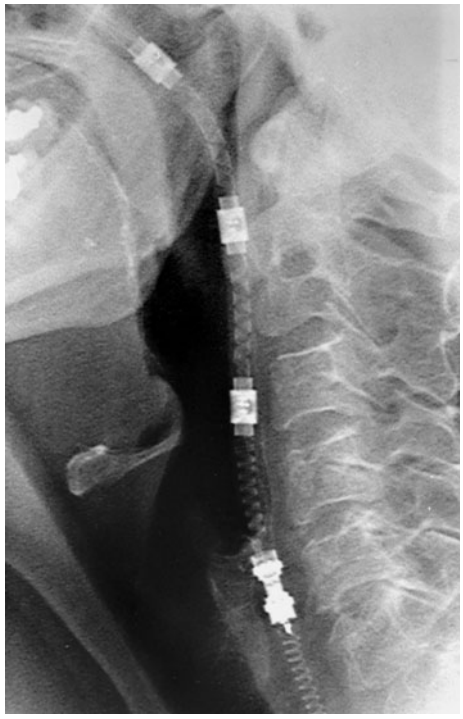

Fig 4.3.1. Pharyngeal manometry with a solid state manometry catheter in the pharynx. The distal sensor is positioned in the upper esophageal sphincter

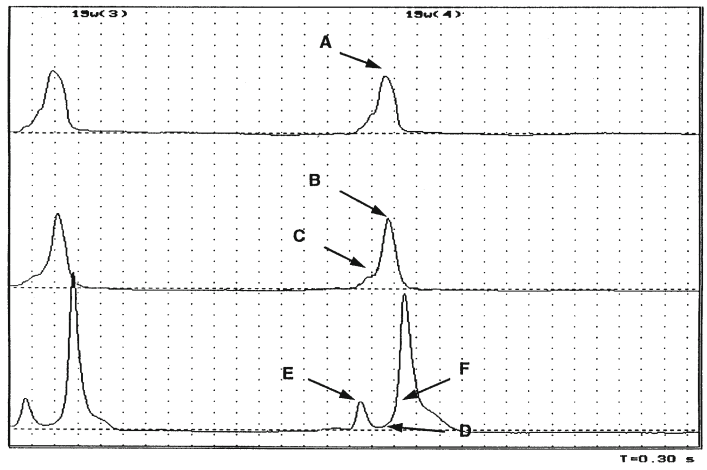

Fig 4.3.2. A manometric tracing from a normal pharyngeal swallow. *A*, pharyngeal peak pressure at the tongue-base level; *B*, pharyngeal peak pressure at the level of the inferior pharyngeal constrictor; *C*, intrabolus pressure; *D*, UES relaxation pressure; *E,F*, UES relaxation duration

Furthermore, the mechanical recording pens of a polygraph record only to a frequency of 20–40 Hz (Stef et al. 1974). Thus, a computerized recording system is needed to faithfully register frequencies up to 48 Hz.

In upper esophageal sphincter manometry, displacement of the manometric sensor is a problem due to the movement of the anatomical structures during swallowing. The Dent sleeve (Dent 1976) is a 6-cm long sensor developed to measure the sphincter pressure in spite of sphincter movement. The sleeve is, however, perfused and the recording accuracy is not in line with solid-state technique. Another drawback with the sleeve is the risk of underestimating sphincter relaxation duration due to the oncoming peristaltic contraction at the top of the sleeve.

We have also studied the movements of the soft palate and the larynx, which are crucial in pharyngeal manometry because of the potential risk of manometry sensor dislocation (Olsson et al. 1994a). A total of 20 dysphagic patients and 20 non-dysphagic volunteers were examined with simultaneous videoradiography and intraluminal pharyngeal solid-state manometry.

The movements of the manometric sensors were analysed from lateral video recording. Two different types of catheter movement were found. The sensor in the upper esophageal sphincter could either be lifted cranially during the closure of the soft palate (type 1) or stay unaltered in the sphincter until the beginning of the laryngeal elevation and then follow the sphincter cranially during laryngeal elevation with no previous response to soft palate closure (type 2). Type 1 movement was found in eight out of 20 patients but in only one of the 20 volunteers. The resting pressure of the upper esophageal sphincter was significantly higher in type 2 (p=0.004). A total of 19 out of 20 participants with a high resting pressure of the upper esophageal sphincter (above 83 mm Hg) were found to have the type 2 movement.

4.3.5. Contact and Cavity Pressures in the Pharynx

Contact pressure is defined when the manometric sensor is in direct contact with the pharyngeal wall with direct contact force on the sensor membrane. Such contact pressure is relatively easy to define and measure, as long as the recording device is properly calibrated and meets the previously described requirements of pharyngeal manometry (Brasseur 1987; Brasseur and Dodds 1991).

Cavity pressures are defined when the manometric sensor is completely surrounded by air or fluid. When the sensor is surrounded by air, for example in the oropharynx, the pressure should be equal to atmospheric pressure, if proper calibration is done. The pressure should then be zero (0 mm Hg).

Another cavity pressure occurs when the sensor is completely surrounded by fluid when swallowing. This pressure is called hydrodynamic pressure or synonymously intrabolus pressure. In a static fluid the pressure is the same at all levels. The pressure in a static fluid will increase with gravity, that means a pressure increase proportional to the depth of the sensor in the fluid. The density of the fluid will also affect the static intrabolus pressure.

In a fluid with flow, however, many different factors become important. This type of intrabolus pressure is very difficult to define and standardise in an exact and reproducible way. The cause of the flow will be a pressure gradient from one place to another within the fluid. The frictional resistance and the fluid acceleration are important. Furthermore, the elasticity of the walls and the viscosity of the fluid has to be considered.

The viscosity can be explained as the friction within the fluid. The units are Ns/m^2 or cp (centipoise) where 1 $cp=1\times10^{-3}$ Ns/m^2. The density of a fluid is also important ($\rho=kg/m^3$).

The rheological properties of the fluid in terms of turbulent or laminar flow are also important.

In a laboratory model we have shown that the intrabolus pressure was dependent on position of the manometric sensor, degree of lumen narrowing, bolus volume, flow rate and fluid viscosity (Olsson et al. 1994b).

We have also elucidated elevated intrabolus pressure above a cricopharyngeal narrowing in a study of 16 patients with cricopharyngeal (CP) bars compared to a control group of 16 patients without CP bars (Olsson and Ekberg 1995). The patients with CP bars showed significantly wider PES (pharyngoesophageal segment) above and below the cricopharyngeal muscle and the contraction pressure within the inferior pharyngeal constrictor was significantly weaker in patients with CP bars. These findings suggested that the cricopharyngeal indentation is due to weak constrictors with outpouching of the gullet above and below the cricopharyngeal muscle. The cricopharyngeal muscle showed no abnormalities in terms of resting pressure, relaxation and contraction pressure. Furthermore there was no significant difference in intrabolus pressure, neither above nor at the level of the cricopharyngeal muscle.

4.3.6. Simultaneous Examination

The combination of simultaneous videoradiography and intraluminal manometry provides fluoroscopic control of sensor positioning and allows for pressure recording and analysis with respect to bolus transport. It combines movement analysis with pressure recordings.

When solid-state sensors are used the technique can be performed in an upright, physiologic position with the patient comfortably seated without the discomfort of swallowing supine and without the discomfort of the continuous flow of perfused manometry (Fig 4.3.3).

The history of simultaneous examinations goes back to 1955 when Fyke and Code measured pharyngeal pressure with a micro-tip transducer fitted on a gastric tube. He used simultaneous cineradiography but the manometric recordings were made with a galvanometer and a polygraph. In 1957 Atkinson et al. took rapid serial radiographs during pharyngeal manometry with perfused technique. In 1966 Sokol and colleagues reported their experience with simultaneous cineradiography and perfused intraluminal manometry of the pharynx.

In 1985 Isberg et al. reported solid-state manometry with the Gaeltec sensors and simultaneous cineradiography. They studied movement of the UES and the manometric device in nine healthy volunteers. They found an upward movement of the manometry catheter that correlated to the elevation of the soft palate. The pressure was recorded on a polygraph.

In 1987 Mendelsohn and McConnel described a simultaneous technique with perfused manom-

Fig 4.3.3. Simultaneous videoradiography and solid-state intraluminal manometry during barium swallow. The pressure registration is displayed on the video screen and also registered on the computer. The subject is comfortably seated and fluoroscopy is performed in a lateral projection

etry and in 1988 McConnel and also Cerenko et al. (1989) described simultaneous fluoroscopy and solid-state manometry. McConnel called the technique manofluorography.

In 1989 JACOB et al. used solid-state manometry and simultaneous videoradiography with polygraph recording to study the UES in eight volunteers. This was a thorough study of UES opening and volume dependent variables such as intrabolus pressure and opening duration.

Also in 1989 Cook et al. described the simultaneous technique in two studies involving healthy volunteers. They used solid-state sensors but a conventional eight-channel polygraph. This was a study of the upper esophageal sphincter where they concluded that UES opening involves sphincter relaxation, anterior laryngeal traction and intrabolus pressure. They also found volume dependent changes in UES dimensions and UES timing indicating a sensory feedback mechanism generated by the brain stem swallow centres.

In 1990 Dantas et al. from the same research group as Cook used the technique in six patients with cricopharyngeal bars. They found normal contact pressures in the pharynx but increased upstream intrabolus pressure. They have also studied different bolus variables with this technique (Dantas 1989).

A high-density barium preparation increased the intrabolus pressure as well as the UES opening duration and anterior hyoid movement. All these studies used the Gaeltec solid-state catheter but recorded on an ordinary polygraph without computerized analysis.

In 1992 Shaker et al. used the perfused sleeve device concurrent with videoradiography to study the esophagoglottal closure reflex. They concluded that esophageal distension by air or balloon evokes a glottal closure mechanism suggesting the existence of an esophagoglottal reflex.

In 1993 Ren et al. used perfused manometry concurrent with videofluoroscopy in a study of intrabolus pressure and concluded that intrabolus pressure can serve as an indicator of the forces resisting peristaltic transport. They called the technique videofluoromanometry. In 1993 Ergun et al. used the simultaneous technique in eight volunteers. They argued strongly for the use of fluoroscopic sensor positioning when performing pharyngeal timing studies. They used solid-state sensors and video recording but polygraph registration without computer. In 1994 Dejaeger et al. used manofluorography to analyse swallowing in the elderly. This study used solid-state technique and video recording but also a conventional polygraph.

In an other study we examined 19 patients (12 males, seven females, mean age 47, range 19–69) with pharyngeal dysphagia but a normal barium swallow and compared them to normal volunteers (Olsson et al. 1995). The patient group showed statistically significant differences for eight out of ten manometric variables.

We found manometric abnormalities that might contribute to dysphagia: five with high UES resting pressure, five with high UES residual pressure, three with weak pharyngeal contractions, seven with prolonged contraction/relaxation time and seven with incoordination.

The conclusion of this study was that manometry could provide additional information in the diagnosis of dysphagia patients.

We have also studied patients with penetration of barium into the larynx during swallowing (Olsson et al. 1998). Videomanometry revealed an increased frequency of manometric abnormalities in patients with penetration. There was however no specific finding and a multitude of abnormalities was found with no association between manometric pathology and the degree of barium penetration.

In patients with pharyngeal retention we found a significantly lower laryngeal elevation indicating the importance of pharyngeal shortening in the swallowing sequence (Olsson et al. 1997).

4.3.7. Conclusion

Simultaneous videoradiography and pharyngeal solid state manometry provides qualitative and quantitative information by assessing bolus transport and intraluminal pharyngeal pressures combined. It can be performed with the subject seated in an upright physiologic swallowing position during fluoroscopy in a gastrointestinal radiology laboratory.

Our experience is that the most complete understanding of bolus transport requires both manometric and radiographic input and that concurrent radiography and manometry will play a more prominent role in the future.

References

Arndorfer RC, Stef JJ, Dodds WJ, Linehan JH, Hogan WJ (1977) Improved infusion system for intraluminal esophageal manometry. Gastroenterology 73:23–27

Ask P, Öberg Å, Tibbling L (1979) Frequency content of esophageal peristaltic pressure. Am J Physiol 236:E296–E300

Atkinson M, Kramer P, Wyman SM, Ingelfinger FJ (1957) The dynamics of swallowing. I. Normal pharyngeal mechanisms. J Clin Invest 36:581–588

Bosma JF, Donner MW, Tanaka E, Robertson D (1986) Anatomy of the pharynx, pertinent to swallowing. Dysphagia 1:23–33

Brasseur JG (1987) A fluid mechanical perspective on esophageal bolus transport. Dysphagia 2:32–39

Brasseur JG, Dodds WJ (1991) Interpretation of intraluminal manometric measurements in terms of swallowing mechanics. Dysphagia 6:100–119

Buchholz DW, Bosma JF, Donner MW (1985) Adaptation, compensation, and decompensation of the pharyngeal swallow. Gastrointest Radiol 10:235–239

Castell JA, Castell DO (1993) Modern solid state computerized manometry of the pharyngoesophageal segment. Dysphagia 8:270–275

Castell JA, Dalton CB, Castell DO (1990) Pharyngeal and upper esophageal manometry in humans. Am J Physiol 258:G173–G178

Castell JA, Castell DO, Schultz AR, Georgeson S (1993) Effect of head position on the dynamics of the upper esophageal sphincter and pharynx. Dysphagia 8:1–6

Cerenko D, McConnel FMS, Jackson RT (1989) Quantitative assessment of pharyngeal bolus driving forces. Otolaryngol Head Neck Surg 100:57–63

Cook IJ, Dodds WJ, Dantas RO, Kern MK, Massey BT, Shaker R, Hogan WJ (1989) Timing of videofluoroscopic, manometric events, and bolus transit during the oral and pharyngeal phases of swallowing. Dysphagia 4:8–15

Dantas RO, Dodds WJ, Massey BT, Kern MK (1989) The effect of high- vs. low-density barium preparations on the quantitative features of swallowing. AJR 153:1191–1195

Dantas RO, Cook IJ, Dodds WJ, Kern MK, Lang IM, Brasseur JG (1990) Biomechanics of cricopharyngeal bars. Gastroenterology 99:1269–1274

Dejaeger E, Pelemans W, Bibau G, Ponette E (1994) Manofluorographic analysis of swallowing in the elderly. Dysphagia 9:156–161

Dent J (1976) A new technique for continuos sphincter pressure measurement. Gastroenterology 71:263–267

Dodds WJ, Logemann JA, Stewart ET (1990) Radiologic assessment of abnormal oral and pharyngeal phases of swallowing. AJR 154:965–974

Ekberg O (1987) Dysfunction of the pharyngo-esophageal segment in patients with normal opening of the upper esophageal sphincter: a cineradiographic study. Br J Radiol 60:637–644

Ekberg O, Nylander G (1982) Cineradiography of the pharyngeal stage of deglutition in 150 individuals without dysphagia. Br J Radiol 55:253–257

Ekberg O, Wahlgren O (1985) Dysfunction of pharyngeal swallowing. A cineradiographic investigation in 854 dysphagial patients. Acta Radiol Diagn 26:389–395

Ergun GA, Kahrilas PJ, Logemann JA (1993) Interpretation of pharyngeal manometric recordings: limitations and variability. Dis Esophagus 6:11–16

Fyke FE, Code CF (1955) Resting and deglutition pressures in the pharyngo-esophageal region. Gastroenterology 29: 24–34

Gordon C, Hewer RL, Wade DT (1987) Dysphagia in acute stroke. BMJ 295:411–414

Groher ME, Bukatman R (1986) The prevalence of swallowing disorders in two teaching hospitals. Dysphagia 1:3–6

Isberg A, Nilsson ME, Schiratzki H (1985) Movement of the upper esophageal sphincter and a manometric device during deglutition. Acta Radiol Diagn 26:381–388

Jacob P, Kahrilas PJ, Logemann JA, Shah V, Ha T (1989) Upper esophageal sphincter opening and modulation during swallowing. Gastroenterology 97:1469–1478

Kronecker H, Meltzer S (1883) Der Schluckmechanismus, seine Erregung und seine Hemmung. Arch Anat Phys (Suppl) 7: 328

Lindgren S, Janzon L (1991) Prevalence of swallowing complaints and clinical findings among 50–79-year-old men and women in an urban population. Dysphagia 6:187–192

McConnel FMS, Cerenko D, Jackson RT, Hersh T (1988) Clinical application of the manofluorogram. Laryngoscope 98: 705–711

Mendelsohn MS, McConnel FMS (1987) Function in the pharyngoesophageal segment. Laryngoscope 97:483–489

Miller AJ (1986) Neurophysiological basis of swallowing. Dysphagia 1:91–100

Olsson R, Ekberg O (1995) Videomanometry of the pharynx in dysphagic patients with a posterior cricopharyngeal indentation. Acad Radiol 2:597–601

Olsson R, Nilsson H, Ekberg O (1994a) Pharyngeal solid state manometry catheter movement during swallowing. A simultaneous videoradiographic and manometric study in 20 dysphagic patients and 20 nondysphagic volunteers. Acad Radiol 1:339–344

Olsson R, Nilsson H, Ekberg O (1994b) An experimental manometric study simulating upper esophageal sphincter narrowing. Invest Radiol 29:630–635

Olsson R, Nilsson H, Ekberg O (1995a) Simultaneous videoradiography and pharyngeal solid state manometry in 25 nondysphagic volunteers. Dysphagia 10:36–41

Olsson R, Castell JA, Castell DO, Ekberg O (1995b) Solid-state computerized manometry improves diagnostic yield in patients with pharyngeal dysphagia: simultaneous videoradiography and manometry in dysphagia patients with normal barium swallows. Abdom Imaging 20:230–235

Olsson R, Castell J, Johnston B, Ekberg O, Castell DO (1997) Combined videomanometric identification of abnormalities related to pharyngeal retention. Acad Radiol 4:349–354

Olsson R, Castell J, Ekberg O, Castell DO (1998) Videomanometry of the pharynx in dysphagic patients with laryngeal barium penetration during swallowing. Acta Radiol 39: 405–409

Orlowski J, Dodds WJ, Linehan JH, Dent J, Hogan WJ, Arndorfer RC (1982) Requirements for accurate manometric recording of pharyngeal and esophageal peristaltic pressure waves. Invest Radiol 17:567–572

Ren J, Massey BT, Dodds WJ, Kern MK, Brasseur JG, Shaker R, Harrington SS, Hogan WJ, Arndorfer RC (1993) Determi-

nants of intrabolus pressure during esophageal peristaltic bolus transport. Am J Physiol 264:G407–G413
Shaker R, Dodds WJ, Ren J, Hogan WJ, Arndorfer RC (1992) Esophagoglottal closure reflex: a mechanism of airway protection. Gastroenterology 102:857–861
Sokol EM, Heitman P, Wolf BS, Cohen BR (1966) Simultaneous cineradiographic and manometric study of the pharynx, hypopharynx, and cervical esophagus. Gastroenterology 51:960–973
Stef JJ, Dodds WJ, Hogan WJ, Linehan JH, Stewart ET (1974) Intraluminal esophageal manometry: an analysis of variables affecting recording fidelity of peristaltic pressures. Gastroenterology 67:221–230
Welch RW, Luckmann K, Ricks PM, Drake ST, Gates GA (1979) Manometry of the normal upper esophageal sphincter and its alterations in laryngectomy. J Clin Invest 63:1036–1041
Winans CS (1972) The pharyngoesophageal closure mechanism: a manometric study. Gastroenterology 63:768–777

4.4 The Post-operative Pharynx and Larynx

Anita Wuttge-Hannig and Christian Hannig

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4.4.1 Introduction

Considerable progress has been made in the past few years in the diagnosis and treatment of swallowing disorders of neurological, anatomic or vascular origin. The treatment has been expanded to include patients with cancer and others with a rather limited time prognosis (Blitzer 1985; Cantarella 1998; Denk et al. 1997; Groher 1992; Hannig and Wuttge-Hannig 1987, 1999; Hannig et al. 1989; Wuttge-Hannig and Hannig 1999; Lazarus et al. 2000; Leonard et al. 2001; Logemann et al. 1994).

This chapter will deal with patients who have undergone ear, nose and throat (ENT) surgery, minor or extensive, and/or radio-, chemo- and the more recent use of radioimmunotherapy and gamma-knife therapy etc., including the sequelae of therapy (Eisbruch et al. 2002; Furia et al. 2000).

The development in recent years of newer diagnostic and therapeutic modalities, including combined surgical and chemotherapeutic and radiotherapeutic schemes resulting in cure or remission even in advanced tumour stages, prompted the urgency to be aware and recognise swallowing complications before sequelae such as aspiration pneumonia become apparent (Hannig et al. 1995a, 1991; Jung and Adams 1980; Walther et al. 1990).

The differentiation between anatomical causes and functional origins related to sensory disturbances which often occur a year or two following the original treatment requires precise analysis in order to initiate appropriate therapy (Hannig and Wuttge-Hannig 1999; Wuttge-Hannig and Hannig 1995). It is also important to exclude submucosal spread of tumour, which may escape endoscopic detection (Wuttge-Hannig et al. 2001). Late consequences of subcutaneous and muscular fibrosis may occur resulting in the restriction of the antero-superior movement of the larynx during swallowing (Hannig 1995).

4.4.2 Altered Anatomy Following Laryngectomy

Dysphagia is often the result of the altered anatomy following surgery such as laryngectomy, whereby separation of the larynx from the anterior pharyngeal wall produces a so-called pharyngeal tube seen especially in wider resections of pharyngeal structures. The configuration and diameter of the pharyngeal tube can vary with different surgical and sewing methods producing wide morphological variations in the radiological appearance (Hannig et al. 1994, 1996; Hannig 1995; Jung and Adams 1980; Martin et al. 1993) (Fig 4.4.1).

The medical literature reports 15%–20% of dysphagia in partial and total laryngectomized patients

A. Wuttge-Hannig, MD
Gemeinschaftspraxis für Radiologie, Strahlentherapie und Nuklearmedizin, Dres. Wuttge–Hannig–Rosskopf–Schepp–Sindelar, Karlsplatz 3–5, 80335 Munich, Germany
C. Hannig, MD
Institut für Röntgendiagnostik des Klinikums rechts der Isar, Technische Universität München, Ismaningerstrasse 22, 82756 Munich, Germany

Fig. 4.4.1. Normal post-surgical anatomy of the pharyngeal tube after total laryngectomy. The pseudoglottis is seen as a circular narrowing

Fig. 4.4.2. Laryngectomy. Tumour recurrence with stenosis of the pharyngeal tube

(Di Santis et al. 1983; Hannig 1995). In our own, 312 patients treated for laryngeal cancer, 37% complained of dysphagia and 19% of an annoying globus sensation. The higher incidence in our patients is probably due to stricter pre-selection of our interdisciplinary group, a heightened awareness as well as better patient education and compliance.

Post-therapeutic dysphagia following laryngectomy may be caused by the following pathology:

1) Tumour recurrence (Fig. 4.4.2)
2) Scarring and benign stenosis (Fig. 4.4.3)
3) Functional disorder of the "pharyngeal tube" and the pharyngo-esophageal transit zone (pseudoglottis) (Fig. 4.4.4)

Functional disorders are difficult to diagnose with video-endoscopy or conventional radiological procedures but require observation of the dynamics of swallowing and analysis of the altered motility of the post-surgical laryngo-pharynx. This is the only way to differentiate between functional changes due to tumour recurrence or scarring.

4.4.2.1 Adapted Physiology of Swallowing

During the pharyngeal phase of swallowing, the force of the dorsal and lateral wave, the counterbalance of the larynx, the tongue-base combined with the occlusion of the soft palate maintain the pressure needed to compress and move the bolus of food. The anatomic loss of soft tissue material causes important disturbances in the propulsion of the bolus (Dodds et al. 1975; Gates 1980; Gay et al. 1984; Hannig 1995). Surgical resection of part of the tongue-base or floor of the mouth is a frequent cause of poor bolus preparation and compression in the oral cavity, thus hindering the trigger for an adequate swallowing reflex. The normal swallowing reflex is effected by an "input summation" needed to reach a critical

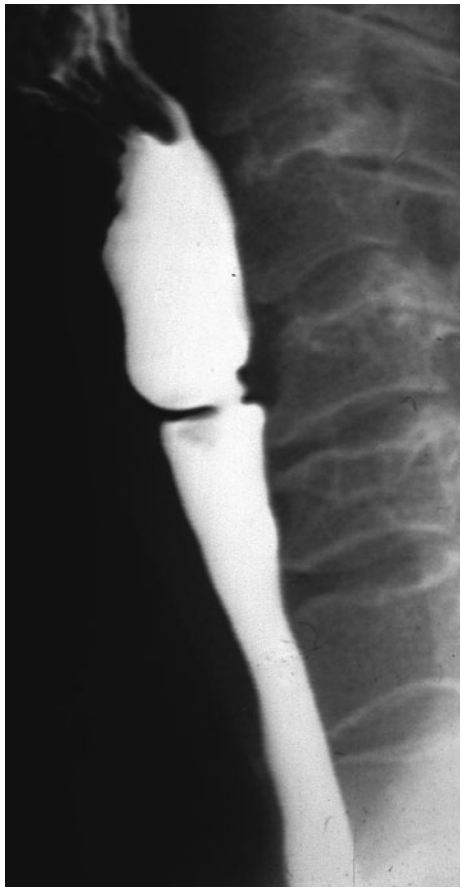


Fig. 4.4.3. Web due to scarring after radiation-therapy

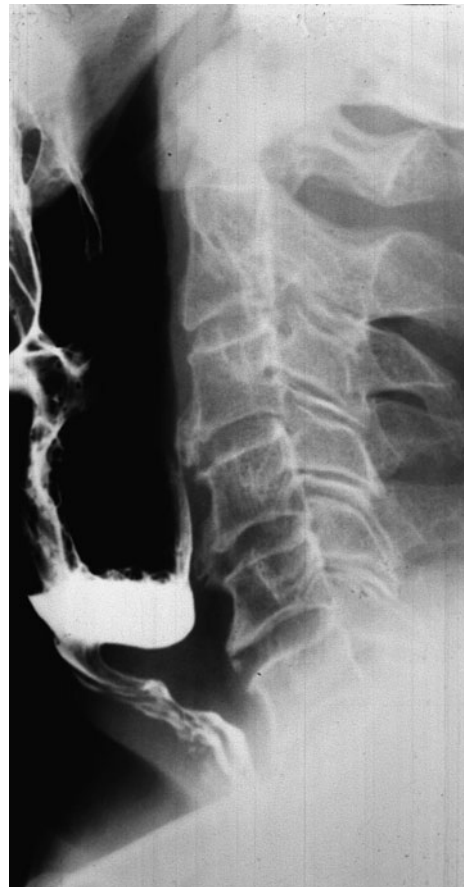


Fig. 4.4.4. Laryngectomy. Hypertrophic pseudoglottis with obstruction of the bolus transport

limit (Kennedy and Kent 1988). This limit is rarely reached in patients treated for an oral-cavity carcinoma, therefore delaying the trigger in the secondary (valleculae) or tertiary area (piriform sinuses) or even quaternary area (laryngeal vestibule). Mucositis and mucosal atrophy due to radiation treatment can also cause trigger delay or absence. The restoration of the mucosal tissue integrity in the pharynx may restore the normal trigger but is frequently incomplete because of the rather permanent xerostomia. It may take 2–3 years of post-radiological treatment for restoration of the integrity of the mucosa. After combined radio-chemo treatment it may never be restored. In the latter patients, lesions in the trigger system occur in the receptors rather than the afferent limb of the loop (Fig. 4.4.5).

Children with a cleft palate present a special group of patients (Bosma et al. 1966; Isberg et al. 1990; Ren et al. 1993; Rubesin et al. 1987) (Fig. 4.4.6). The maturation of the swallowing reflex, which in nursing

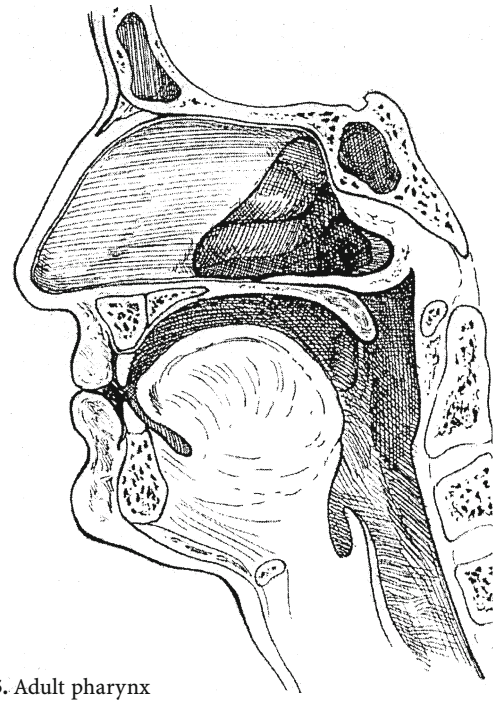


Fig. 4.4.5. Adult pharynx

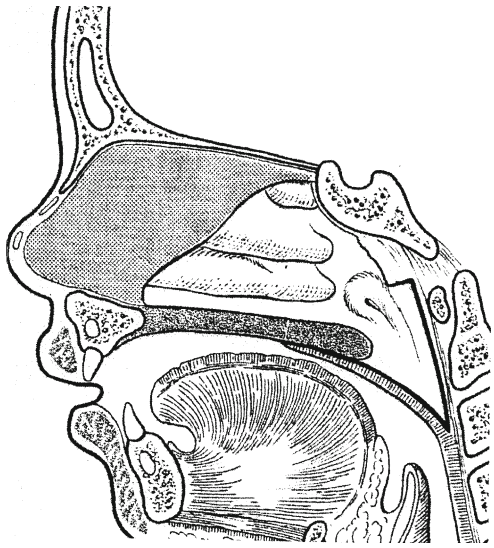


Fig. 4.4.6. Infantile pharynx with short distance between the base of the tongue and the laryngeal vestibule

children occurs in the secondary area (the valleculae), cannot be brought up in time to the first area as is the case in adults, namely the arch of the fauces and dorsal pharyngeal wall, because the pressure generation in the oropharynx is inadequate. The lack of counter-balance of the soft palate during swallowing may cause a compensatory downward movement by the tongue-base in order to propagate the bolus into the pharynx.

A unilateral incision of the pharynx causes likewise lack of propagation and transport of the bolus retaining part of the bolus in the ipsilateral vallecula and piriform sinus. The most serious motility change occurs as a result of resection of the larynx and the resulting motility changes of the "neopharynx" referred to as the "pharyngeal tube" (Fig. 4.4.7). In such patients even a hypertonic dorsal wave is incapable of producing a competent peristaltic wave. The pharyngeal pressure generation is not sufficient to passively open the upper esophageal sphincter (UES). We demonstrated that in patients with a hypotonic wave and a normal UES at least 40 mm Hg manometric pressure is needed for the passive opening of the dissected pharyngo-esophageal segment.

4.4.2.2 Adaptation Methodology

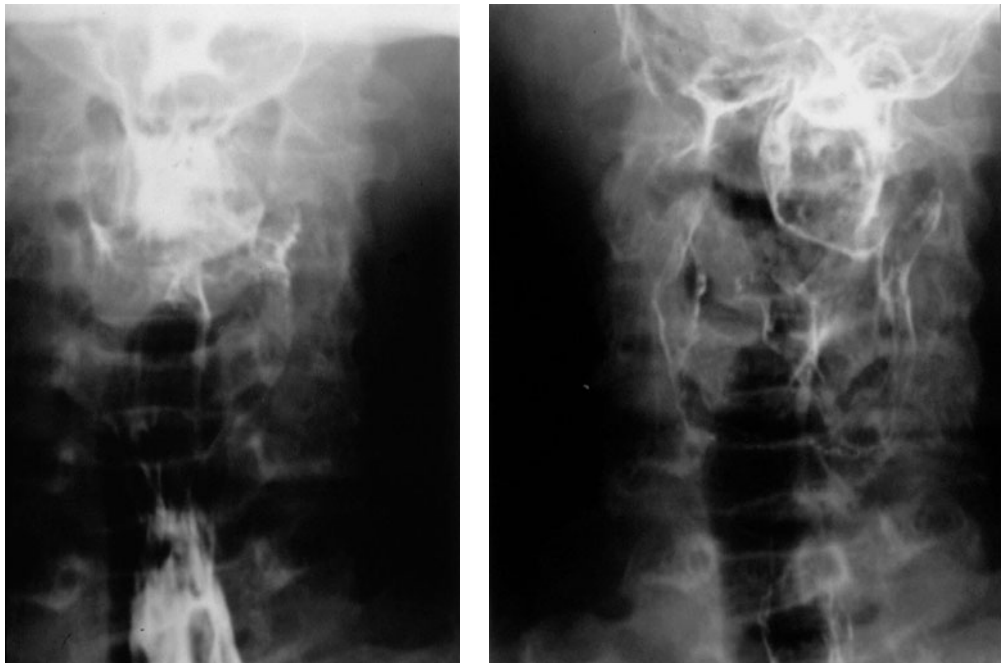
In our swallowing-impaired patients we use an isoosmolar iodine solution (Isovist® -Iotrolan which causes no lung damage in case of aspiration. (The hyperosmolar preparations may cause pulmonary oedema) (Hannig et al. 1994; Hannig 1995). The contrast medium is mixed with the type of food that causes the swallowing difficulty. This can be solid, semi-solid, crumbly or liquid.

In patients after surgery for a cleft palate or following partial or complete laryngectomy we also have the patient perform different sounds and vocals, such as "coca-cola", "in the garden grows an apple tree", "kakadu". The elasticity of the pharynx and larynx is tested by the use of Valsalva (Fig. 4.4.8) and Mueller manoeuvres producing a maximal distension or collapse of the pharynx. This allows us to check for an occult neoplastic process of scarring.

4.4.2.3 Morphological and Functional Swallowing Abnormalities

A wide variety of morphological and functional swallowing abnormalities can be seen following ENT neoplasm surgery. The causes vary with the different types of surgery. Oral, pharyngeal, laryngeal and thyroid neoplasms are treated with a variety of procedures ranging from laser excision, wide resection, horizontal and transverse pharyngeal resection, partial or total laryngectomy or thyroidectomy, including combinations of therapy with or without hyper-

Fig. 4.4.7. Laryngectomy. Motor disturbance of the dorsal pharyngeal wall with diverticulum between the middle and inferior constrictors



a b

Fig. 4.4.8a,b. Oro-hypopharyngeal neoplasm of the posterior wall best demonstrated by Valsalva manoeuvre

thermia present with different forms of dysphagia. Post-therapeutic changes related to vascular surgery of the neck, combinations of chemo- and radiation treatment may also result in dysphagia.

4.4.2.4 Laryngectomy

The incidence of dysphagia and globus pharyngis following total laryngectomy is around 50% and rises with additional radiotherapy. Radiotherapy alone causes less of the latter but causes more frequently loss of sensation resulting in "trigger" delay. The displacement of the larynx results in loss of compression of the anterior pharyngeal wall, resulting in a hypercompetent dorsal wave of the reformed pharyngeal tube. Lateral compression during peristalsis is also deficient. The propulsion force is accomplished by the rotation of the tongue-base which can result in hyperexcursion compensating for the dorsal pharyngeal wall (Hannig et al. 1994; Jung and Adams 1980; Mills 1973; Pauloski et al. 2002). A too large resection of the tongue-base may cause severe dysphagia. A deficient opening of the reconstructed pharyngeal wall and the sphincter sleeve results in dysphagia. Hypotonicity of the pharyngeal tube may prevent the passive opening of the reconstructed sphincter. A hyperactive sphincter could also be responsible for solid state dysphagia. Since voice prosthesis users form sounds with the reconstructed sphincter sling, a perfect voice quality correlates frequently with the degree of dysphagia. Our own patients favoured sacrificing the discomfort for a better voice quality (Hannig et al. 1994, 1993a).

Another cause of dysphagia is displacement of a voice prosthesis, particularly a too high implanted or a too steeply inclined prosthesis, causing food impaction and both solid and liquid dysphagia. Table 4.4.1 lists the changes in the area of the UES and pharynx in relation to the associated clinical symptoms. In one third of patients the dysphagia could be related to functional disorders. Post-radiation and post-therapeutic scar stenosis or membra-

Table 4.4.1. Dysphagia present in laryngectomie

	causing dysphagia	not causing dysphagia
Delayed opening of the UES	51%	49%
Premature closure of the UES	65%	35%
Incomplete opening of UES	70%	30%
Hypopharyngeal diverticula	52%	48%
Web	54%	46%
Reduced contractility/scarring of the pharyngeal tube	80%	20%
Tumor recurrence	31%	69%

(n = 246, male : female = 77% : 23%, median age 61,5 aa)

Table 4.4.2. Dysphagia present in laryngectomie alterations in pharyngeal timing in relation to dysphagia

	causing dysphagia	not causing dysphagia	normal value
Time of pharyngeal peristalsis	1047 ms	763 ms	617 ms
Time of pharyngeal passage	868 ms	721 ms	615 ms
Depth of constriction wave (quotient)	0,60	0,69	0,47

(n = 246, male : female = 77% : 23%, median age 61,5 aa)

nous stenosis is quite frequent but a recurrence is rare. In two thirds of our patients with dysphagia, dysfunction of the motility of the pharynx or the UES could be identified. In asymptomatic patients slight dysmotility of the UES is still seen but is much less than in the symptomatic patients. The obstruction during a delayed opening of the UES was less than 20% in those patients. UES function was assessed using planimetry and frame-by-frame analysis. The median delay caused by the dysmotility of the UES was 47.5 ms, considerably longer than the 24.5 ms in the asymptomatic patients. Two thirds of our symptomatic patients had dysphagia and one third only globus sensation. We were interested to see that there was a correlation between the extent of swallowing symptoms and the hypercontraction of the neopharynx. Table 4.4.2 shows the median values of time of peristalsis, the time of pharyngeal passage and the constrictor ratio (maximal protrusion of the dorsal pharyngeal wave in relation to the sagittal diameter of the C3 vertebral body). The median obstruction of the area of the pharyngo-esophageal segment due to a dysfunction of the sphincter was 35% in the symptomatic group. It is remarkable how the time of peristalsis and time of pharyngeal passage was absolutely abnormal in the dysphagic patients. In Table 4.4.2, the motility disorders of the pharyngeal tube and the UES are presented in relation to the time measurements. As expected, due to the altered anatomy after laryngectomy, the constriction ratio was quite high in both the symptomatic and asymptomatic patient. Due to the loss of the laryngeal counter-pressure, the dorsal peristaltic wave gets unusually prominent. In addition to the dysfunction of the neoglottis (formed by the UES) we see a second constricted area in the cervical esophagus just below the UES. This "second sphincter" showed a functional pattern comparable to the UES. The exact pathomechanism of this second hyper-contractile zone is yet unknown. It might be the effect of regulation of the airflow during the esophageal voice production.

In a large number of patients with a delayed emptying of the pharynx due to insufficient peristalsis we were able to observe a marked compensation by the tongue-base, a virtual "tongue pump" (Hannig 1995).

This tongue pump consists of an accentuated caudo-dorsal movement of the floor of the mouth together with the tongue base moving the bolus through the pharynx and the UES. In some patients an additional hyperexcursion of the Passavant cushion and the region of the superior constrictor pharyngis was noted (a temporary long contact of the dorsal pharyngeal wall and tongue-base). Quite often, scarring resulted in reduced contractility of the reconstructed pharynx or of a narrow pharyngeal canal. In Figure 4.4.9 the scar was caused by radiotherapy. In other patients it could be the result

Fig. 4.4.9. Supraglottic pharyngo-laryngeal resection

of chronic inflammation or in a poor healing of a post-operative fistula.

4.4.2.5 Hemi-pharyngectomy

There are two main types of partial pharyngeal resections. The horizontal resection (Fig. 4.4.9) is uncommon today, due to long-term complications from the interruption of the constrictor muscles resulting in deficient bolus propulsion. The more common longitudinal resection includes resection of the unilateral vallecula and piriform sinus and reconstruction of the pharyngeal tube by means of suturing the dorsal and anterior pharyngeal wall, often in combination with a myocutaneous flap. This reconstructed pharyngeal structure does not participate in the normal constriction due to relative weakness of the muscles. The bolus often remains on the resected side and can be cleared out of the pharynx only through the healthy hemi-pharynx (Hannig and Wuttge-Hannig 1999; Hannig 1995).

Fig. 4.4.10. Fistulation to the residual tongue-base after partial tongue resection and radiochemotherapy

4.4.2.6 Post-therapeutic Pharyngeal Cancer

In cancers of the oral cavity, the floor of the mouth, tongue, tonsils, pharynx and UES, the morphological and functional changes correspond to the type and extent of therapy (Hannig and Wuttge-Hannig 1999; Rosen et al. 2001; Zuydam et al. 2000). This may include simple resection, additional radio- or combined radio-chemotherapy. Less frequent forms of therapy such as combinations with hyperthermia are rare and will not be considered in our comparative studies (Fig. 4.4.10).

Table 4.4.3 summarises the functional disorders. Combined radiation and chemotherapy and surgery alone present similar problems, whereas radiation therapy alone presents the least. The most severe problems occur when surgery is followed by radiotherapy whereby in addition to the trigger problem related to the surgery, additional fibrosis related to the radiotherapy occurs. Differences in the severity and type of aspiration related to the different treatment modalities are illustrated in Table 4.4.4. Aspiration turns out to be most frequent in radiotherapy in combination with chemotherapy in which the negative effects of receptor destruction, hypostimulation and mucosal damage are added to the radiation and chemotherapy combination. Nevertheless, surgery alone causes the highest incidence

Table 4.4.3. Functional Alterations due to ENT Cancer-Therapy

	Surg n=37	Rth n=47	Surg+Rth n=92	Rth+Chemo n=145
Bolus formation	46%	11%	87%	25%
Premature leaking out of the oral cavity	85%	37%	77%	44%
Delayed trigger of the swallowing reflex	49%	78%	88%	81%
Penetration in the laryngeal vestibule	40%	19%	76%	72%
Aspiration	75%	71%	79%	75%
Reduced coughing reflex	–	6%	32%	8%
Laryngeal antero-cranial- movement	24%	22%	73%	43%
Dysfunction of the UES	65%	9%	86%	56%

n = 321, m:f = 67:33, mean age = 55,7 aa

Table 4.4.4. Type and Severity of aspiration due to ENT Cancer-Therapy

	Surg 27/32 82%	Rth 36/45 80%	Surg+Rth 78/92 79%	Rth+Chemo 112/145 77%
Predeglutitive	16	24	36	47
Grade I	4	9	10	21
Grade II	4	6	9	9
Grade III	6	5	9	9
Grade IV	2	4	8	8
Intradeglutitive	3	0	13	21
Grade I	–	–	2	3
Grade II	2	–	4	7
Grade III	1	–	3	9
Grade IV	–	–	4	2
Postdeglutitive	8	12	29	44
Grade I	2	3	12	15
Grade II	2	2	9	13
Grade III	4	5	5	10
Grade IV	–	2	3	6
Combined forms	15	7	51	49

n = 321, m:f = 67:33, mean age = 55,7 aa

of aspiration followed by radiotherapy alone and surgery plus radiotherapy. The high number of aspiration complications related to radiotherapy alone could be due to the fact that it usually involves older patients who could not tolerate a combined protocol. Table 4.4.5 lists the morphological alterations relative to the therapeutic protocol. As expected radiotherapy alone, surgery alone, and radio-chemotherapy cause less problems.

4.4.2.7 Cleft Palate

A palatal cleft causes social stigma due to the external aesthetic image, the palato-rhinolalia and the nasal penetration of the bolus during swallowing (Engelke et al. 1991; Engelke 1994; Hannig et al. 1993b). The anatomical shortening and the functional restriction of the soft palate include the post-therapeutic scar after closure of the defect. The above can lead to a velo-pharyngeal insufficiency.

An analysis of the velo-pharyngeal closing mechanism should be done before and after surgery (Thumfart et al. 1996; Bergeron et al. 1984; Croft et al. 1981; Dalston et al. 1985; Engelke et al. 1991; Engelke 1994; Hannig and Wuttge-Hannig 1992; Hannig et al. 1993b, 1995b; Hartmann et al. 1972; Hess et al. 1994, 1995a, 1995b, 1996a, 1996b; Herzog et al. 1993). The early use of surgical occlusion and/or prosthesis may help achieve proper maturation from the infantile to the adult swallowing reflex pattern. The nursing infant adapts safely to the anatomical changes. The first segment of the nearly horizontal aditus laryngis, which remains very narrow to the base of the tongue, together with the large valleculae and the long soft palate, permits a two-chamber separation of the pharynx. The infant is thus able to fill up the valleculae with usually three suctions during breathing. Only after three stimulations of the tongue base can the swallowing reflex, located very deep in the pharynx, be triggered. Mastication assists in the development of the orally directed swallowing reflex. Tactile stimulations are also helpful in this process. According to Croft et al. (1981), four types of velo-pharyngeal occlusion mechanisms can be identified: sagittal, coronal, circular and circular with a Passavant cushion (Fig. 4.4.11).

The commonly used tools are: lateral radiography of the cranium, a pantogram of the dental arcade, video-fluoroscopy and the 16-slice helical CT already separated in spatial but not temporal resolution in the electron beam tomogram. In the future, MRI will be the imaging modality of choice especially in infants and young adults in whom the radiation dose is critical and where many radiograms in different

Table 4.4.5. Morphologic Alterations due to ENT Cancer-Therapy

	Surg n=20	Rth n=47	Surg+Rth n=92	Rth+Chemo n=145
Fibrosis of the subepidermal tissue	11%	62%	72%	48%
Fibrosis of muscles	–	52%	82%	58%
Adhesions skin-larynx	15%	49%	74%	48%
Adhesions larynx-prevertebral fascia	–	48%	72%	27%
Stenosis by scarring	12%	42%	66%	53%
Oral defects	78%	–	58%	18%
Pharyngeal defects	29%	11%	61%	7%
Laryngeal defects	32%	–	48%	–
Recurrence	–	7%	11%	21%

n = 321, m:f = 67:33, mean age = 55,7 aa

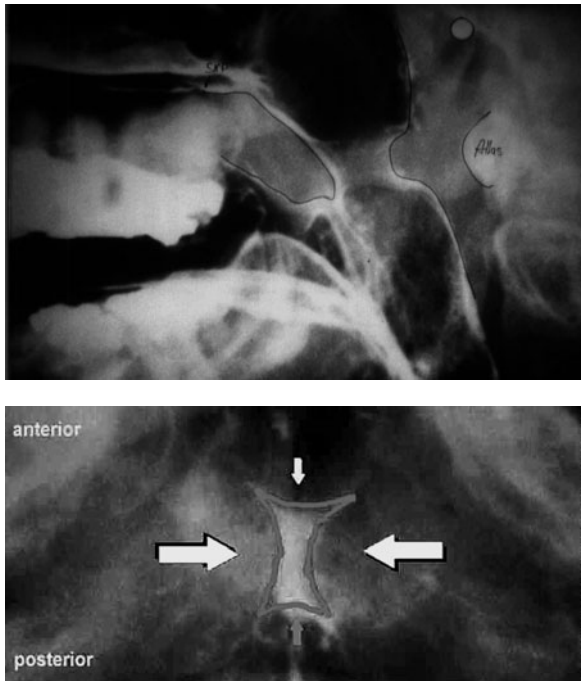


Fig. 4.4.11a,b. Cleft. **a** Insufficient nasopharyngeal closure with attempt of a Passavant's compensation. **b** Circular compensation with major action of the lateral pharyngeal constrictors in hemiaxial projection

for such patients. Therapy is planned based on dynamic swallowing analysis. A division and grading analysis into pre-, intra-, and post-deglutitive aspiration and its severity is done. The accurate study of the multiple patho-mechanisms allows us to design a program individually tailored for each patient, which gives the best results and is most cost-effective (Hannig et al. 1995a; Hannig and Wuttge-Hannig 1999).

A unilateral functional defect or hypotonicity suggests a unilateral cerebral event whereas a bilateral defect suggests a neuromuscular disorder (Wuttge-Hannig and Hannig 1995). Dilatation of the hypopharynx is common in trumpet players and singers and is bilateral. The study can identify neurogenic, myogenic and reflux induced disorders. The reflux episodes could be due to the tenth cranial nerve. The differentiation is important since in myogenic dysfunction simple drug use might solve the problem, which is not the case in vagal dysfunction where not only the UES but also the esophageal clearing function is compromised. A myotomy of the UES should only be performed in rare cases. Esophago-pharyngeal and tracheal aspiration would result from a surgical dilatation of the PE segment. The passive opening of the UES can be influenced by planes have to be used. A very accurate appreciation of the velum motility and the swallowing competence allows one to decide which therapeutic approach would be most favourable: simple augmentation of the dorsal wall (Hynes) or the more complicated velo-pharyngoplasty (Sanvenero-Roselli) (Horch et al. 1993; Sader et al. 1994, 1995, 1997, 2001; Skolnick et al. 1989). Post-therapeutic evaluation of the velum performance assists in the decision whether a re-operation or a conservative program is to be pursued.

4.4.3 Post-traumatic, Post-lesional and Post-surgical Brain Lesions

A large spectrum of damage to the CNS and peripheral nervous system can result in dysphagia (Hannig 1995; Wuttge-Hannig and Hannig 1995). In neurologically impaired patients, conservative rehabilitative methods are the procedures of choice. Surgery might cause further damage to the trigger area. The Munich rehabilitative strategy is generally adopted

Fig. 4.4.12. Laryngo-hyoido-mentopexy in a patient with an intradeglutitive aspiration due to a resection of an ependymoma of the IVth ventricle

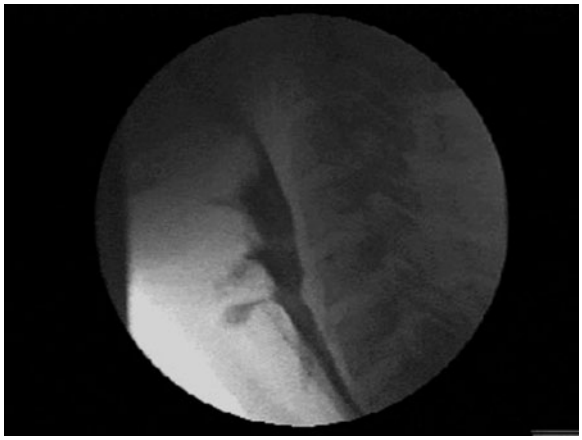
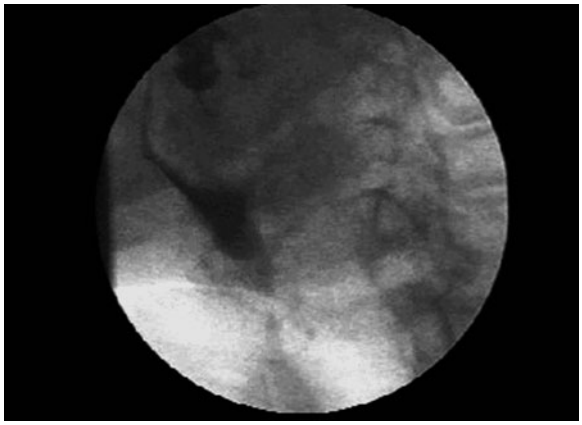


Fig. 4.4.13. Patient after surgery and radiotherapy for a thyroid cancer 25 years previously. Fistulation below the level of the arytenoid cartilages

a



b

Fig. 4.4.14a,b. Patient with dysphagia due to a patch insertion in the right carotid artery. **a** By turning the head to the left side the bolus passage is obstructed. **b** By turning the head to the right side a good clearance of the pharynx can be achieved

the Mendelsohn manoeuvre but if there is a spasm of the UES conservative therapy is rarely adequate. Further reconstructive surgery should be undertaken only after a long-term rehabilitative trial. In the latter patient the separation of the alimentary and respiratory canal should only be a last choice. If the patient possesses good cognitive capacity the use of alternative positional manoeuvres could be used. This is especially important in the planning of a laryngo-hyoido-mentopexy procedure planned via dynamic imaging. This surgical technique necessitates deglutition in the upright position (Hannig 1995). In 1996 Mahieu described a similar method as "laryngeal suspension", which was performed in a large number of aspirating patients but which excluded the tracheal stabilisation (Fig. 4.4.12). ENT and neurologists use botulism toxin injections. Newer applications of the latter may cure esophageal motility disorders causing dysphagia. Within Thumfart's group in Innsbruck and at the ENT departments in Turin and Milan botox injections are frequently used (Cantarella 1998; Pastore 1997; Thumfart et al. 1996).

4.4.4 Scarring and Post-surgical Instabilities of the Pharynx

Dysphagia is quite frequent as a result of muscular dissection in vascular (Fig. 4.4.13) and orthopaedic surgery of the cervical spine area (Hannig and Wuttge-Hannig 1999; Hannig 1995). In surgical unilateral procedures an out-pouching of the weaker hemi-pharynx may occur due to a less effective lateral constrictor wave. The therapeutic approach in such cases depends on the extent of weakness of the unilateral pharynx. If there is a relatively weak pharyngeal wall the bolus will end up in this weak hemi-pharynx and will not be cleared out of it. If there is a scar or hypertonicity of the surgical side, the contralateral side will transport the bolus well. A trial of the best manoeuvre by means of dynamic imaging will yield the best results. The raising or inclination of the head may result in a different pre-load to the external laryngeal musculature. The swallowing disorder may get even more complex by adhesions of the skin of the neck to the larynx frequently after surgery for thyroid tumours (Fig. 4.4.14).

4.4.5 Conclusion

Dynamic imaging in swallowing disorders defines the pathophysiology of the disorder and determines the method of rehabilitation of the post-therapeutically impaired deglutition. Myo- or neurogenic disorders can be identified. The oral and pharyngo-esophageal interaction can be studied. The advantage of this method of study lies in its ability to produce the high spatial and temporal resolution of the very fast motility between the oral cavity and entrance into the esophagus. This procedure may also provide hints as to the localisation of the neurological focus, although the nature of the underlying disease is rarely identified by this technique alone. The studies permit individual tailoring of rehabilitation and the most cost-effective method.

References

Bergeron RT, Osborn AG, Sam PM (1984) Head and neck imaging. CV Mosby, St Louis, USA

Blitzer A (1985) Evaluation and management of chronic aspiration. Am Arch ENT

Bosma JF, Truby HM, Lind J (1966) Distortions of the upper respiratory and swallowing motions in infant having anomalies of the upper pharynx. Acta Paediatr Scand 163:111–128

Cantarella G (1998) Definizione ed epidemiologia dei disturbi della deglutizione. Centro Richerce e Sudi Amplifon, Raccolta bibliografica Seminario Turbe della deglutizione: attualità diagnostiche e terapeutiche: 12–17

Croft CB et al. (1981) Patterns of velopharyngeal valving in normal and cleft palate subjects: A multi view videofluoroscopic and nasoendoscopic study. Laryngoscope 91:265–271

Dalston RM et al. (1985) The diagnosis of velopharyngeal insufficiency. Clin Plastic Surg 12:685–695

Denk DM, Swoboda H, Schima W, Eibenberger K (1997) Prognostic factors for swallowing rehabilitation following head and neck cancer surgery. Acta Otolaryngol 117: 769–774

Di Santis DJ, Balfe DM, Koehler RE, Lee KT (1983) Barium examination on the pharynx after vertical hemilaryngectomy. AJR 141:335–339

Dodds WJ, Hogan WJ, Lynden SB, Stewart ET, Stef JJ, Arndorfer RC (1975) Quantitation of pharyngeal motor function in normal human subjects. J Appl Physiol 39:692–696

Eisbruch A, Lyden T, Bradford CR, Dawson LA, Haxer MJ, Miller AE, Teknos TN, Chepeha DB, Hogikyan ND, Terrell JE, Wolf GT (2002) Objective assessment of swallowing dysfunction and aspiration after radiation concurrent with chemotherapy for head-and-neck cancer. Int J Radiati Oncol Biol Phys 53:23–28

Engelke W (1994) Videoendoskopische Untersuchungen des velopharyngealen Sphinkters bei Gesunden und bei Gaumenspaltpatienten. Dtsch Z Mund Kiefer Gesichtschir 18:190–195

Engelke W, et al. (1991) Elektromagnetische Artikulographie: Eine neue Methode zur Untersuchung von Bewegungsfunktionen des Gaumensegels. Folia Phoniatr 43: 147–152

Furia CL, Carrara-de Angelis E, Martins NM, Barros AP, Carneiro B, Kowalski LP (2000) Videofluoroscopic evaluation after glossectomy. Arch Otolaryngol Head Neck Surg 126: 378–383

Gates GA (1980) Upper esophageal sphincter: pre- and postlaryngectomy – a normative study. Laryngoscope 90:454–464

Gay I, Crisin R, Elidan J (1984) Myotomy of the cricopharyngeal muscle. A treatment for dysphagia and aspiration in neurological disorders. Rev Laryngol Otol Rhinol (Bord) 105:271–274

Groher M (1992) Dysphagia – diagnosis and management, 2nd edn. Butterworth-Heinemann, Boston

Hannig C (1995) Functional fluoroscopic diagnosis of pharynx and esophagus Springer, Berlin

Hannig C, Wuttge-Hannig A (1987) Stellenwert der Hochfrequenzkinematographie in der Diagnostik des Pharynx und Ösophagus. Röntgenpraxis 40:358–377

Hannig C, Wuttge-Hannig A (1992) Cineradiography in pre- and postoperative evaluation of cleft patients. 6th Annual Meeting of the European Society of Head and Neck Radiology. Karlsruhe, Oct. 7–10, 1992

Hannig C, Wuttge-Hannig A (1999) Radiological functional diagnosis of swallowing disorders in neuological diseases and in posttherapeutic oncological ENT-diseases. In: Bartologme G, Buchholz DW, Feussner H, Hannig C, Neumann S, Prosiegel M, Schöter-Morasch H, Wuttge-Hannig A (eds) Schluckstörungen Diagnostik und Rehabilitation Hrsg, 2. Auflage. Urban und Fischer Verlag, Germany, pp 65–110

Hannig C, Wuttge-Hannig A, Hörmann M, Herrmann IF (1989) Kinematographische Untersuchungen des Pathomechanismus der Aspirationspneumonie. ROFO Fortschr Geb Rontgenstr Nuklearmed 150:260–267

Hannig C, Wuttge-Hannig A, Clasen B, Kellermann S, Volkmer C (1991) Dysphagia of the treated laryngeal cancer – detection of functional and morphological changes by cineradiography. Bildgebung Imaging 58:141–145

Hannig C, Feussner H, Stein H (1993a) Cineradiography and radiomanometry in the pre- and postoperative evaluation of cricopharyngeal dysfunction. Scientific Programme and Abstracts, ECR 93, Springer International 1993:111

Hannig C, Wuttge-Hannig A, Daschner H, Sader R (1993b) Pre- and postoperative evaluation of cleft-patients by cineradiographic imaging. Scientific Programme and Abstracts, ECR 93. Springer International 273

Hannig C, Stein H, Wuttge-Hannig A, Hess U (1994) Diagnosis of cricopharyngeal dysfunction using simultaneous videomanometry and cinemanometry. 3rd Annual Dysphagia Research Society Meeting, Oct 14–16, 1994 McLean, Virginia, USA

Hannig C, Hess U, Sader R, Sinz J (1995a) Einfluss des velopharyngealen Abschlusses auf die OP-planung. Symposium „Moderne Chirurgi der Kieferfehlstellungen". Klinik und Poliklinik für Mund-Kiefer-Gesichtschirurgie, Technische Universität München, März 24–25, 1995

Hannig C, Hess U, Wuttge-Hannig A, Volkmer C (1996) Dys-

phagie nach Laryngektomie – morphologische versus funktionelle Veränderungen. ROFO Fortschr Geb Rontgenstr Nuklearmed Suppl 164:95

Hannig C, Wuttge-Hannig A, Hess U (1995b) Analyse und radiologisches Staging des Typs und Schweregrades einer Aspiration. Der Radiologe 35:741–746

Hartmann H et al. (1972) Zur Frage der Intelligenz und sozialen Entwicklung von Kindern mit Lippen-, Kiefer-Gaumenspalten. Prax Kinderpsychol Kinderpsychiatr 21: 1–10

Herzog M et al. (1993) Röntgenbefunde nach sekundärer bzw tertiärer Osteoplastik bei Lippen-Kiefer-Gaumen-Spalten. Fortschr Kiefer Gesichtschir 38:64–66

Hess U, Hannig C, Sader R, Cavallaro A, Wuttge-Hannig A, Zeilhofer H (1994) Assessment of the velopharyngeal closure with high-frequence-cineradiography for preoperative planning of maxillary advancement, June 15–19, 1994, ICHNR, Washington DC

Hess U, Hannig C, Weiss W, Sader R, Zeilhofer H, Wuttge-Hannig A, Merl T (1995a) Die Videokinematographie in der prä- und postoperativen Diagnostik der Lippen-Kiefer-Gaumen-Spalte. Radiologe 35:712–715

Hess U, Hannig C, Weiss W, Sader R, Zeilhofer HF, Wuttge-Hannig A, Merl T (1995b) Die Videokinematographie in der prä- und postoperativen Diagnostik der Lippen-Kiefer-Gaumenspalten. Radiologe 35:712–715

Hess U, Hannig C, Cavallaro A, Sader R (1996a) Die postoperative Kontrolle der seskundären Velopharyngoplastik mit Hilfe der Kinematographie. 77. Deutscher Röntgenkongress, Mai 15–18, 1996, Wiesbaden. ROFO Fortschr Geb Rontgenstr Nuklearmed 164:95

Hess U, Hannig C, Sader R, Cavallaro A, Wuttge-Hannig A, Zeilhofer H (1996b) Die Bewertung des velopharyngealen Verschlusses zur präoperativen Planung der Oberkiefervorverlagerung. Röntgenpraxis 49:25–26

Horch HH et al. (1993) Klinische Ergebnisse nack sekundärer Kieferspaltosteoplastik im Wechselgebiss für Lippen-Kiefer-Gaumenspalten. Fortschr Kiefer Gesichtschir 38: 61–64

Isberg A et al. (1990) Radiographic examination of velopharyngeal function. In: Delbaso A (ed) Maxillofacial imaging. Saunders, Philadelphia

Jung TK, Adams GL (1980) Dysphagia in laryngectomised patients. Otolaryngol Head Neck Surg 88:25–33

Kennedy JG, Kent RD (1988) Physiological substrates of normal deglutition. Dysphagia 3:24–38

Lazarus CL, Logemann JA, Pauloski BR, Rademaker AW, Larson CR, Mittal BB, Pierce M (2000) Swallowing and tongue function treatment for oral and oropharyngeal cancer. J Speech Lang Hear Res 43:1011–1023

Leonard JR, Kendall KA, Johnson R, McKenzie S (2001) Swallowing in myotonic muscular dystrophy: a videofluoroscopic study. NY State Dent J 82:979–985

Logemann JA, Bytell DE (1979) Swallowing disorders in three types of head and neck surgical patients. Cancer 44:1095–1105

Logemann JA, Gibbons P, Rademaker AW, Pauloski BR, Kahrilas PJ, Bacon M, Bowman J, McCracken E (1994) Mechanism of recovery of swallow after supraglottic laryngectomy. J Speech Hear Res 37:965–974

Mahieu HF (1996) Aspiration in the late pharyngeal phase: UES dysfunction or defective laryngeal mobility? 2nd International Symposium on Laryngeal and Tracheal Reconstruction, Mai 22–26, 1996, Monte Carlo Proceedings 33

Martin BJ, Schleicher MA, O´Connor A (1993) Management of dysphagia following supraglottic laryngectomy. Clin Commun Disord 3:27–36

Mills CP (1973) Dysphagia in pharyngeal paralysis treated by cricopharyngeal sphincterotomy. Lancet 1:455–457

Pastore A, Marchese Ragona R, De Grandis D (1997) Il ruolo della tossina botulinica nelle turbe dello sfintere esofageo superiore. Corso intensivo teorico-pratico: Diagnostica e terapia die disturbi della deglutitzione. (Abstract book pp 20–25) Milano 30–31.10.1997.

Pauloski BR, Rademaker AW, Logemann JA, Lazarus CL, Newman L, Hamner A, MacCracken E, Gaziano J, Stachowiak L (2002) Swallow function and perception of dysphagia in patients with head and neck cancer. Head Neck 24:555–565

Ren YF, Isberg A, Henningsson G (1993) Interactive influence of a pharyngeal flap and adenoid on maxillofacial growth in cleft lip and palate patients. Cleft Palate Craniofac J 30:144–149

Rosen A, Rhee TH, Kaufman R (2001) Prediction of aspiration in patients with newly diagnosed untreated advanced head and neck cancer. Arch Otolaryngol Head Neck Surg 127:975–979

Rubesin SE, Jones B, Donner MW (1987) Radiology of the adult soft palate. Dysphagia 2:8–17

Sader R, Horch HH, Herzog M, Zeilhofer HF, Hannig C, Hess U, Bünte E, Böhme G (1994) Hochfrequenz-Videokinematographie zur objektiven Darstellung des velopharyngealen Verschlussmechanismus bei Gaumenspaltenpatienten. Fortschr Kieferorthop 55:169–175

Sader R, Horch HH, Zeilhofer HF, Deppe H, Hannig C, Hess U (1995) High-frequency cineradiography for objective three dimensional rendering of velopharyngeal closure in cleft patients. In: Functional surgery of the head and neck. Kärcher H (ed) RM-Druck u Verl, Graz 31–36

Sader R, Zeilhofer HF, Horch HH (1997) Maxillary advancement and velopharyngeal closure in cleft patients. In: Lee ST, Huang M (eds) Transaction 8th International Congress on Cleft Palate and Related Anomalies, Stamford Press Pte Ltd, Singapore, pp 651–654

Sader R, Zeilhofer HF, Dietz M, Bressmann T, Hannig C, Putz R, Horch HH (2001) Levatorplasty, a new technique to treat hypernasality: anatomical investigations and preliminary clinical results. J Craniomaxillofac Surg 29: 143–149

Skolnick ML et al. (1989) Videofluoroscopic studies of speech in patients with cleft palate. Springer, Berlin Heidelberg New York

Thumfarth WF, Potoschnig C, Dapunt U, Nekahm D (1996) Diagnostic and surgical systems for management of aspiration and swallowing disturbances. 2nd International Symposium on Laryngeal and Tracheal Reconstruction, Monte Carlo Proceedings 31

Walther EK, Rodel R, Deroover M (1990) Rehabilitation of deglutition in patients with pharyngeal carcinoma. Laryngorhinootologie 69:360–368

Wuttge-Hannig A, Hannig C (1995) Radiologische Differentialdiagnose neurologisch bedingter Schluckstörungen. Der Radiologe 35:733–740

Wuttge-Hannig A, Hannig C (1999) Anatomy of swallowing In: Bartologme G, Buchholz DW, Feussner H, Hannig C,

Neumann S, Prosiegel M, Schöter-Morasch H, Wuttge-Hannig A (eds) Schluckstörungen Diagnostik und Rehabilitation Hrsg. (2. Auflage) Urban und Fischer Verlag, Germany, pp 1–11

Wuttge-Hannig A, Beer A, Gebhardt A, Hellerhoff P, Wuttge R, Hannig C (2001) Alternative methods for the diagnostic of deglutition. In: Schindler O, Ruoppolo G, Schindler A (eds) Deglutologie. Omega Edizioni,

Zuydam AC, Rogers SN, Brown JS, Vaughan ED, Magennis R (2000) Swallowing rehabilitation after oro-pharyngeal resection for squamous cell carcinoma. Br J Oral Maxillofac Surg 38:513–518

4.5 The Therapeutic Swallowing Study

Margareta Bülow and Bonnie Martin-Harris

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4.5.1 Introduction

When selecting an instrumental procedure to assess oral and pharyngeal swallowing dysfunction videoradiography is considered the gold standard and is the most frequently used technique (Donner 1988; Jones and Donner 1989; Ekberg 1990, 1992; Dodds et al. 1990a, 1990b). The videofluoroscopic swallowing study is also referred to as the modified barium swallow study and has been shown to have high clinical yield (Martin-Harris et al. 2000). Because of the therapeutic implications of the swallowing study, as well as the therapeutic approaches that can be applied and tested during the examination, the study will be described as "the therapeutic swallowing study" in this chapter. The therapeutic swallowing study permits observation of upper aerodigestive tract function as the patient swallows varied volumes and textures of different radiopaque materials (Logemann 1986) and requires specialized training for accurate and reliable implementation and interpretation of test results (Logemann et al. 2000). The study is recorded on a dynamic medium such as videotape or computer disk making it possible to analyse structural movements in relation to contrast flow in slow motion and frame by frame. Pathology can be identified such as disordered timing and coordination of structural movement, and the presence, degree, timing and cause of aspiration can be documented.

The therapeutic swallowing study is a dynamic procedure that examines the mechanical passage of food and liquid from the mouth to the stomach. However, in most studies the focus lies on oral and pharyngeal phases of deglutition. Comprehensive examination includes the observation of oral bolus manipulation, lingual motility efficiency of mastication, timing of pharyngeal swallow initiation, soft palate elevation and retraction, tongue base retraction, pharyngeal contraction, superior and anterior hyolaryngeal movement, epiglottic inversion and extent and duration of pharyngoesophageal segment opening (Martin-Harris et al. 2000).

Various therapeutic strategies, such as postural techniques, manoeuvres and techniques improving oral sensory awareness can be systematically applied, tested and their effect on function can be observed (Ekberg 1986; Logemann et al. 1989; Martin et al. 1993; Martin 1994; Bülow et al. 2001). Different materials are given to the patient to identify optimal food and liquid textures that facilitate a safe and efficient oral intake.

Clinical experience and research findings provide evidence that aspiration, and more importantly the cause of aspiration, can be missed during observations made from test swallows included in a clinical or bedside examination. Studies have shown that clinicians do not consistently identify the presence of aspiration during clinical examinations. The sensitivity and specificity of the bedside examina-

M. Bülow, SLP, MSc
Neurological Department & Radiological Department, Malmö University Hospital, SE 205 02 Malmö, Sweden
B. Martin-Harris, PhD, CCC-SLP
Director, MUSC Evelyn Trammell Institute for Voice and Swallowing Disorders, Otolaryngology Head and Neck Surgery, Medical University of South Carolina, Charleston, South Carolina, USA

tion in the detection of aspiration and in predicting patient outcome warrants further study. Furthermore, it has been reported that 50%–60% of patients who aspirate do not cough (Linden and Siebens 1983; Splaingard et al. 1988; De Pippo et al. 1992; Nathadwarawala et al. 1992, 1994; Zenner et al. 1995). Despite its limitations, the bedside or non-instrumental examination provides important information regarding signs and symptoms of potential swallowing disorders and need for further instrumental examination, impressions regarding the patient's language and cognitive status, propensity for fatigue during eating and drinking, and a realistic picture of the patient's eating and drinking patterns.

Fig 4.5.1. The laboratory

4.5.2 How to Perform the Study

At the radiological department at the University Hospital MAS, Malmö, Sweden, we have had nearly ten years experience with a swallowing assessment team that includes collaboration between a speech/language pathologist and radiologist in the performance and analysis of the therapeutic swallowing study. The speech/language pathologist has her own schedule two half days a week at the laboratory and schedules her own patients. The teamwork of the two specialists provides rapid and adequate information about current swallowing dysfunction and management recommendations to the referring clinician, patient and caregivers.

The swallowing recording equipment includes:

- Fluoroscope
- Time-date character generator
- SVHS videotape recorder which have jog and shuttle and frame lock abilities to enable observations in slow motion
- Microphone
- Video monitor for SVHS quality

The patient is administered controlled food and liquid textures that are mixed with contrast resulting in a simulated diet but dense enough to allow X-ray visualization. Typically, barium sulfate is used and allows for optimal visualization of bolus passage through the alimentary tract (Murray 1999).

The procedure is most often performed with the patient seated in an upright position. If the individual is unable to assume and maintain a seating position, adaptive seating devices can be employed. If theses devices are not available, the patient may be placed on the fluoroscopic table and the examination is performed with the patient lying down. The head and trunk can then be raised to a semi-upright position. The individual can also be positioned seated in his or her own wheelchair if there is adequate distance between the floor and fluoroscopic tube to permit oro-pharyngeal and cervical esophageal viewing.

The study typically begins with the subject in lateral view, the optimal position for visualizing penetration or aspiration of material into the laryngeal vestibule before, during, and after swallowing. In lateral view the profile contours of the soft palate, base of the tongue, posterior pharyngeal wall, epiglottis, aryepiglottic folds, anterior hypo-pharyngeal wall and the region of the cricopharyngeal muscle or pharyngoesophageal segment. Following swallowing assessment in the lateral view, the patient is then positioned in a frontal view permitting assessment regarding asymmetric contours, the surface of the base of the tongue, median and lateral glossoepiglottic folds, tonsillar fossa, valleculae and hypopharynx.

In our experience the amount of radiation during the study is low, 2–5 mSv (absorbed dose), which is about one eighth of the amount when performing a colon examination. The average radiation exposure time is 2–3 min.

It is critical that caregivers, nurses (e.g., depending on the medical status of the patient) and/or family members observe the study either at the time of the examination or at a later viewing of the recorded examination. This provides the opportunity to educate the caregivers in the nature of the patient's swallowing problem, and the necessary precautions and management strategies that must be applied to ensure airway protection and efficient oral intake.

4.5.3 Routines During the Study

The swallowing therapist:

- Decides how the procedure will be performed based on observations of the patient during the bedside examination. The volumes and textures of contrast materials used in the study should be tailored to meet the particular needs of the patient based on his/her clinical swallowing presentation.
- Selects and prepares contrast materials (mixing, measured volumes, order).
- Prepares the patient for the examination.
- Provides instructions and administers contrast materials.
- Systematically applies trial therapeutic strategies based on the observed nature of the swallowing disorder.
- Documents a collaborative report with the radiologist that includes recommendations for non-oral intake, appropriate food/liquid textures and bolus volumes, and necessary therapeutic strategies such as compensatory postures, manoeuvres and exercises.

The radiologist:

- Operates the fluoroscopic equipment and observes anatomic abnormalities.
- Documents a collaborative report with the swallowing therapist.

The assistant nurse:

- Prepares the fluoroscopy suite for the study.
- Prepares the appropriate test material.
- Completes the registration and operates the videotape recorder.
- Assists the patient before and after the study.
- Completes the swallowing protocol during the procedure.

4.5.4 Test Material

Every procedure is individually adapted to the patient, even if the same routines are used. If there is a suspicion of aspiration and/or we do not know for sure whether the patient will initiate a pharyngeal swallow, the procedure starts with 2 or 3 ml of water-soluble contrast either as thin or thickened liquid. The patient may not be exposed to any risks of aspiration pneumonia.

The normal procedure consists of the following consistencies:

- Pudding
- Puree
- Chopped solid material: either meat or vegetables in sauce
- Thickened liquid
- Thin liquid

When testing mastication some sort of crisp bread covered with barium paste is used.

Every test material is given three times in various amounts from 3 ml, 5 ml to 10 ml or more. However, few trials may be given depending on the severity of the swallowing impairment and degree of aspiration. The patients may be fed by the swallowing therapist but may also feed themselves, giving important information about their habitual eating and drinking behaviours. For example, patients suffering from right hemispheric damage often take excessive amounts of solids and liquids during mealtime. This is important behaviour to identify and modify. Studies showing typical bolus volume during thin liquid swallows indicate that the average volume habitually ingested by males is 21.3–29.3 ml and 13.6–20.4 ml in females (Adnerhill et al. 1989). If the patient wishes, or if he/she reports symptoms with only very specific food or liquid items, he/she may bring material to the examination where it will be mixed with barium contrast and tested.

The material used is prepared in the hospital kitchen according to our recipe and the radiological department provides the kitchen with barium sulfate. When the material comes to the radiological department in small cans, 0.5 dl for a solid bolus and 1 dl for liquids, it is placed in a freezer. Before every assessment the assistant nurse takes it from the freezer in time for the study. Every patient gets his or her individual combination of test material depending on the nature of the swallowing problem.

Fig 4.5.2. Test material

Table 4.5.1. Recipe of material for the therapeutic swallowing study

1 portion solid bolus consists of 45 g product and 15 g mixobar HB barium contrast and gives 60 g ready-mixed material (0.5 dl)
1 portion thickened liquid consists of 100 g product and 30 g mixobar HB barium contrast and gives 130 g ready mixed material (1 dl)

4.5.5 Protocol I: Therapeutic Videoradiographic Swallowing Examination

Table 4.5.2. Protocol for therapeutic videoradiographic swallowing examination

Date: _____

Us nr: _____

Personnumber:_____Name:_____

Actual problems:_____

Actual nutrition: Oral 100% 50% 25% Non oral: Nasogastric tube PEG Infusion
Actual consistencies: Solid bolus Puree Pudding Other Liquids: Thin liquid Thick liquid

Consistency: Amount: Intake:	1 2 3 ml	5 ml	10 ml	20 ml	Drinks without help	Eats without help	Have to be fed	Syringe
Solid bolus								
Chopped solid bolus								
Purée								
Pudding								
Thin liquid								
Thick liquid								
Water-soluble								
Bread								

Comments:

THERAPEUTIC STRATEGIES:
Sensory stimulation: TTS Push down with a spoon against the tongue Sour bolus (lemon) Larger bolus volume Cold bolus Chewing Postural techniques: Chin tuck Head back Head rotated to damaged side Head rotated Head tilt to stronger side Swallowing techniques: Supraglottic swallow Super-supraglottic swallow Effortful swallow Mendelsohn Manoeuvre

TREATMENT PLAN:

Diet modification:

4.5.6
Protocol II: Therapeutic Videoradiographic Swallowing Examination

Table 4.5.3. Protocol for therapeutic videoradiographic swallowing examination. Radiological Department, University Hospital MAS, Malmö

Examination number: …… Date of examination: ……..

Name: …… Person nr :……..

Medical diagnosis: ……

Remittent:: ……

Exam performed by: ……

CONSISTENCIES:		Thin liquid	Thick liquid	Pudding	Puree	Solid bolus
Lateral view:						
ORAL PREPARATION:	Normal	0	0	0	0	0
	Incomplete lip closure	1	1	1	1	1
	Delayed bolus preparation	2	2	2	2	2
	Diffuse spreading of bolus in oral cavity	2	2	2	2	2
ORAL PHASE:	Normal	0	0	0	0	0
- Leakage	Anterior:	1	1	1	1	1
		2	2	2	2	2
	Posterior:	1	1	1	1	1
		2	2	2	2	2
- Tongue movements	Normal coordination	0	0	0	0	0
	Inefficient	1	1	1	1	1
- Mastication	Normal	0	0	0	0	0
	Inefficient	1	1	1	1	1
- Delayed bolus transport	Mild	1	1	1	1	1
	Moderate	2	2	2	2	2
Regurgitation into nasal cavity		1	1	1	1	1
DISSOCIATION	<0.5 s	0	0	0	0	0
	0.5–2 s	1	1	1	1	1
	3–5 s	2	2	2	2	2
	>5 s	3	3	3	3	3
PHARYNGEAL PHASE:						
- Laryngeal elevation, Movements of os hyoid	Normal	0	0	0	0	0
	Reduced	1	1	1	1	1
	Anterior movement incomplete	2	2	2	2	2
	Elevation absent	3	3	3	3	3
- epiglottic movement	Normal	0	0	0	0	0
	Incomplete	1	1	1	1	1
Vestibulum penetration - aspiration	No	0	0	0	0	0
	Subepiglottic penetration	1	1	1	1	1
	Supraglottic penetration	2	2	2	2	2
	Tracheal penetration	3	3	3	3	3
Constrictor muscles/ Retention	No	0	0	0	0	0
	Weak muscles, +/- mild retention	1	1	1	1	1
	Paresis one segment, +/- moderate retention	2	2	2	2	2
	Severe paresis, +/- severe retention	3	3	3	3	3
PES	Normal opening	0	0	0	0	0
	< 25 % impaired	1	1	1	1	1
	25–50 % impaired	2	2	2	2	2
	> 50 % impaired	3	3	3	3	3
Frontal view:						
Unilateral paresis Bilateral paresis Vocal fold closure						
Comments: Total points:						

4.5.7 Swallowing Management

Upon completion of the therapeutic swallowing study, the swallowing therapist has identified the nature and severity of the swallowing disorder, made recommendations for oral versus non-oral intake, and designs an individual treatment plan directed toward specific swallowing functional outcome goals. Every applied treatment strategy must be based on a sound rationale according to the nature of the swallowing problem and the physical and cognitive status of the patient.

Strategies may include postural techniques, sensory improvement techniques, swallowing manoeuvres, isometric exercises applied to the muscle groups of the tongue and suprahyoid musculature, and bolus volume and texture modification. Combinations of different management strategies are often used.

A critical component in the management of dysphagic patients is providing accurate *information. To inform and explain current problems in an understandable way* is essential for the patient, family members and nursing staff in terms of heightening awareness and understanding of the dysfunction, and highlighting the necessity for swallowing precautions and treatment. The successful rehabilitation of a dysphagic patient will to a large extent depend on the implementation of a team approach, with the patient and family as key components of the team.

Education of the medical and nursing staff regarding how to observe potential swallowing problems, and hosting routine patient care rounds facilitates appropriate patient referrals and expedites the care of dysphagic patients

The Postural Techniques

To change head or body posture in dysphagic patients is for most individuals relatively easy to perform and can successfully eliminate misdirected swallows (penetration/aspiration) on liquids 75%–80% of the time (Logemann 1998). *Head down (chin tuck)* widens the valleculae, pushes the tongue base backward toward the pharyngeal wall, places the epiglottis in a more posterior position and narrows the entrance to the larynx. This position is used in cases where there is a reduction in posterior tongue base motion, unilateral laryngeal dysfunction, delayed initiation of pharyngeal swallow and in reduced laryngeal closure. For patients with weak pharyngeal constrictor muscles, a chin tuck position makes the difficulties worse, especially when swallowing a masticated bolus, which could lead to increased retention in the pharyngeal recess and post-swallow aspiration (Shanahan et al. 1993; Welch et al. 1993; Bülow et al. 1999). But chin tuck could reduce the depth of misdirected swallows (penetration/aspiration) (Bülow et al. 2001).

By using the *head back* posture, gravity can facilitate more efficient clearance of the oral cavity when oral transit is disturbed; however, good airway protection and pharyngeal swallowing mechanics must be present for safe implementation of this posture. When the *head is rotated to the damaged side* the passage through the damaged or weakened side is reduced permitting primary bolus passage through the stronger side. The *head rotation posture also pulls* the cricoid cartilage away from the posterior pharyngeal wall and facilitates passage into the cervical esophagus as in the case of decreased pharyngoesophageal segment opening (Logemann et al. 1989). *Head tilt to the stronger side results in* bolus passage down on the stronger side in *the case of* unilateral oral and pharyngeal weakness. *Lying down on one side* will take advantage of the gravity effect on pharyngeal retention, and reduce the likelihood of aspiration of residue after the swallow in cases of reduced pharyngeal contraction.

Sensory Improvement Techniques

Techniques such as thermal tactile stimulation and bolus manipulation are designed to improve oral sensory awareness and improve the timing of swallow initiation (Lazzara et al. 1986; Rosenbek et al. 1996). Furthermore, bolus could be manipulated in different ways such as giving a sour bolus, a cold bolus, a larger volume bolus and a bolus that requires chewing. Pressure applied to the tongue during spoon administrations of food may also facilitate productive tongue movement toward a functional swallow.

Swallowing Manoeuvres

The four different techniques that are designed to change a selected aspect of the physiology of pharyngeal swallow include: supraglottic swallow, super-supraglottic swallow, effortful swallow and the Mendelsohn manoeuvre (Logemann 1998; Logemann and kahrilas 1990; Kahrilas et al. 1992). Despite their proven effectiveness in some patients, the complexity of the manoeuvre often precludes their usage with patient experiencing language-cognitive impairment, pulmonary disease, deconditioning and fatigue.

Supraglottic swallow and Super-Supraglottic swallow

The primary purpose of these techniques is to ensure airway protection prior to and throughout the swallow.

- The techniques include instructing the patient to: (1) take a breath, hold it and, in the super-supraglottic swallow, bear down; (2) swallow; (3) clear their throat without inhaling; (4) dry swallow. It has been shown that instructing the patient to hold their breath "hard" and "bear-down" results in optimal glottic and supraglottic closure (Martin et al. 1993; Logemann 1998).

The purpose by bearing down in the super-supraglottic swallow technique is to assist the closure of the posterior glottis and, the false vocal folds.

Effortful Swallow

The purpose of this technique is to increase posterior motion of the tongue base during pharyngeal swallow. The increase in tongue base retraction associated with the manoeuvre should facilitate improved bolus clearance from the valleculae (Logemann 1998). This technique could also reduce the depth and severity of misdirected swallows (penetration/aspiration) (Bülow et al. 2001).

Instructions:

- Squeeze hard with all of your tongue muscles when you swallow.

Mendelsohn Manoeuvre

The purpose of this manoeuvre is to increase the duration and extent of laryngeal elevation and thereby increase the width and duration of (pharyngoesophageal) cricopharyngeal opening (Logemann 1998). Following early relaxation of the cricopharyngeus muscle, the pharyngoesophageal segment is pulled open as the cricoid cartilage is moved away from the posterior pharyngeal wall during upward and forward movement of the hyoid bone and larynx. The functional result of this technique is to facilitate bolus passage through the pharyngoesophageal segment and decrease the degree of pyriform residue (Lazarus et al. 1993).

Instructions:

- Pay attention to your neck by swallowing your saliva several times.
- Try to feel how your Adam's apple lifts and lowers as you swallow.
- Swallow again, and when you feel the Adam's apple lift, keep it in its highest position by squeezing the muscles of your tongue and neck for several seconds.

Fig 4.5.3. Pharyngeal dysfunction

Oral Motor Exercises

By performing specialized exercises to the striated musculature of the tongue, pharynx and cervical esophagus region, there is some evidence to suggest that it is possible to improve muscle strength and range of motion (Logemann 1983, 1995; Sonies 1993). The exercise program must be individually adapted depending on the specific type(s) of swallowing impairment, and clearly documented and explained to ensure independent patient implementation whenever possible. Some exercises have prescribed intensities and frequencies, such as the Shaker exercise (Shaker et al. 1997). Other isometric strengthening exercises are usually introduced in a hierarchy of difficulty, with a gradual increase in intensity and frequency. Based on what is known about skeletal muscle physiology in other parts of the body, it is likely that the patient will need to continue an exercise maintenance program even after functional swallowing skills are acquired.

Diet Modification

Food and liquid texture modifications are often found to be necessary based on the results of the therapeutic swallowing study. Examples include changes from thin to thickened liquids, or vice versa, and soft solids to pureed foods. Even though these texture modifications appear to be simply strategies to improve swallow safety and efficiency, several factors must be considered when making these recommendations: (1) patient tolerance and

preference; (2) adequate nutrition and hydration; (3) cultural considerations regarding food items and textures.

Oral versus Non-oral Feeding

Tube feeding methods, such as nasogastric tube, percutaneous endoscopic gastrostomy (PEG) or jejunostomy are sometimes the only safe and efficient avenues for feeding in severely dysphagic patients. It is a common occurrence for a patient to have tube feedings as the primary source of nutrition and hydration, with safe supplementation of small amounts of modified food and liquid textures for pleasure and optimising of quality of life.

The lateral radiograph illustrate a tracheal penetration and residue in valleculae and pyriform sinus secondary to a brain-stem stroke.

4.5.8 Therapeutic Strategies (Table 4.5.4)

Table 4.5.4. Disorders documented on videofluoroscopy and management. [(Modified from Martin (1994)]

Radiographic presentation	Cognitive and sensory stimulation, oral motor exercises, diet modification, alternative nutrition	Head positioning	Manoeuvres
Oral phase: Anterior leakage due to incomplete lip closure Oral residue Posterior leakage due to spill over tongue base Delayed bolus preparation Diffuse spreading of bolus in oral cavity Inefficient tongue movements and mastication Delayed bolus transport Aspiration before pharyngeal swallow Regurgitation into nasal cavity	Optimise liquid/ food texture Sufficient bolus volumes Intraoral placement to unimpaired side Labial resistive exercises Buccal range of motion exercises and resistive exercises Lingual range of motion exercises and resistive exercises Bolus control exercises Thickened liquids, cold liquids, semisolids, soft solids Controlled bolus volume Thickened liquids and semisolids Bolus hold exercises	Head tilt to unimpaired side	Lip pursing Double swallow Supraglottic swallow
Dissociation: Delayed initiation of pharyngeal swallow	Thermal Tactile Stimulation. Thickened liquids. Cold stimulus. Controlled bolus volume Bolus hold exercises	Chin tuck	Supraglottic swallow
Pharyngeal phase: Laryngeal elevation reduced Movements of os hyoid: Elevation incomplete, anterior movement incomplete Epiglottis - incomplete closure Misdirected swallows: Vestibulum laryngis - subepiglottic penetration - supraglottic penetration Tracheal penetration -aspiration Weakness of constrictor muscles: Mild, moderate or severe Retention Unilateral, pocketing in valleculae or/and piriform sinus Bilateral, pocketing in valleculae or/and piriform sinuses	Controlled bolus volume Slightly thickened liquids Thinned semi-solids Cold stimulus Thickened liquids Thermal tactile stimulation Bolus hold exercises Controlled bolus volume Liquids, semisolids and soft solids Hard swallow Controlled bolus volume Liquids and thinned semi-solids	Chin tuck Head rotation toward impaired side Head tilt toward unimpaired side Head rotation	Mendelsohn manoeuvre Mendelsohn manoeuvre Supraglottic swallow Supraglottic swallow Double swallow Modified supraglottic swallow Double swallow Modified supraglottic swallow
PES: - impaired opening Regurgitation from esophagus to piriform sinuses	Liquids, semisolids and soft solids Controlled bolus volumes		Mendelsohn manoeuvre Double swallow Modified supraglottic swallow
Absent pharyngeal swallow	Thermal tactile stimulation Tube feeding.		

4.5.9 Conclusion

In the therapeutic swallowing study a trained speech/language pathologist and a radiologist collaborate in performing the examination. The competence of the two specialists provides an opportunity for a complete visualization and analysis of the entire swallowing sequence. Testing swallowing function with varied bolus volumes and textures, and the implementation of trial therapeutic strategies are integral components of the examination. Despite the strengths and clinical utility of the therapeutic swallowing study, there are several limitations with interpretation of test results across swallowing centres. There is lack of standardization regarding the textures of contrast materials, varying terminology and methods of analysis, and generally suboptimal intersubject reliability regarding the rating of swallowing function. Clearly there is a need for multi-centre collaboration to determine salient features that warrant analysis on the therapeutic swallowing study, standardization of rating scales and the association of these ratings with patient functional outcomes and quality of life. The evaluation of oro-pharyngeal swallowing function is in its infancy, and we expect that evidenced based studies will increase across centres in Europe and the United States to assist us in further determining the optimal test protocols, item analysis and treatment strategies for improved swallowing function in our dysphagic patients.

References

Adnerhill I, Ekberg O, Groher ME (1989) Determining normal bolus size for thin liquids. Dysphagia 4:1

Bülow M, Olsson R, Ekberg O (1999) Videomanometric analysis of supraglottic swallow, effortful swallow and chin tuck in healthy volunteers. Dysphagia 14:67–72

Bülow M, Olsson R, Ekberg O (2001) Videomanometric analysis of supraglottic swallow, effortful swallow and chin tuck in patients with pharyngeal dysfunction. Dysphagia 16: 190–195

De Pippo KL, Holas MA, Reding MJ (1992) Validation of the 3-ounce water swallow test for aspiration following stroke. Arch Neurol 49:1259–1261

Dodds WJ, Logemann JA, Stewart ET (1990a) Radiological assessment of abnormal oral and pharyngeal phases of swallow. AJR Am J Roentgenol 154:965-974

Dodds WJ, Stewart ET, Logemann J (1990b) Physiology and radiology of the normal oral and pharyngeal phases of swallowing. AJR Am J Roentgenol 154:993–963

Donner M (1988) The evaluation of dysphagia by radiography and other methods of imaging. Dysphagia 1:49–50

Ekberg O (1986) Posture of the head and pharyngeal swallow. Acta Radiol Diagn 27:691–696

Ekberg O (1990) The role of radiology in evaluation and treatment of neurologically-impaired patients with dysphagia. J Neurol Rehabil 4:65–73

Ekberg O (1992) Radiologic evaluation of swallowing. In: Groher ME (ed) Dysphagia. Diagnosis and Management, 2nd edn. Butterworth-Heineman, Boston, pp 163–195

Jones B, Donner MW (1989) How I do it: examination of the patient with dysphagia. Dysphagia 4:162–172

Kahrilas PJ, Logemann JA, Gibbons P (1992) Food intake by maneuver: an extreme compensation for impaired swallowing. Dysphagia 7:155–159

Lazarus CL, Logemann JA, Gibbons P (1993) Effects of manoeuvres on swallowing function in a dysphagic oral cancer patient. Head Neck 15:419–424

Lazzara G, Lazarus C, Logemann J (1986) Impact of thermal stimulation on the triggering of the swallow reflex. Dysphagia 1:73–77

Linden P, Sibens A (1983) Dysphagia: Predicting laryngeal penetration. Arch Phys Med Rehabil 64:281–283

Logemann J (1998) Evaluation and treatment of swallowing disorders. Pro-Ed, Austin, Texas

Logemann JA (1983) Evaluation and treatment of swallowing disorders. Pro-Ed, Austin, Texas

Logemann JA (1986) A manual for the videofluoroscopic evaluation of swallowing. College-Hill Press, Boston

Logemann JA (1995) Dysphagia: evaluation and treatment. Folia Phoniatr Logop 47(3):140–164

Logemann JA, Kahrilas PJ (1990) Relearning to swallow post-CVA: application of maneuvers and indirect biofeedback. A case study. Neurology 40:1136–1138

Logemann JA, Kahrilas PJ, Kobara M, Vakil NB (1989) The benefit of head rotation on pharyngoesophageal dysphagia. Arch Phys Med Rehabil 70:767–771

Logemann JA, Lazarus CL, Phillips Keely S, Sanches A, Rademaker AW (2000). Effectiveness of four hours of education in interpretation of radiographic studies. Dysphagia 15: 180–183

Martin BJW (1994) Treatment of dysphagia in adults. In: Reiff Cherney L (ed) Clinical managements of dysphagia in adults and children. Aspen Publishers, Inc. Gaithersburg, Maryland, chap. 6, pp 153–183

Martin BJW, Logemann JA, Shaker R, Dodds WJ (1993) Normal laryngeal valving patterns during three breath-hold manoeuvres: a pilot investigation. Dysphagia, 8:11–20

Martin-Harris B, Logemann JA, McMahon S, Schleicher MA, Sandidge J (2000) Clinical utility of the modified barium swallow. Dysphagia 15: 136–141

Murray J (1999) Videofluoroscopic examination. In: Manual of dysphagia assessment in adults. Singular Publishing Group, Inc. San Diego, London, chap. 3, pp 113–151

Nathadwarawala KM, Nicklin J, Wiles CM (1992) A timed test of swallowing capacity for neurological patients. J Neurol Neurosurg Psych 55:822–825

Nathadwarawala KM, Mc Groary A, Wiles CM (1994) Swallowing in neurological outpatients. use of a timed test. Dysphagia 9:120–129

Rosenbek JC, Roecker EB, Wood JL, Robbins J (1996) Thermal application reduces the duration of stage transition in dysphagia after stroke. Dysphagia 11:225–233

Shaker R, Kern M, Bardan E, Taylor A, Stewart ET, Hoffamnn RG, et al. (1997) Augmentation of deglutitive upper esoph-

ageal sphincter opening in the elderly by exercise. Am J Physiol G1518–G1522

Shanahan TK, Logemann JA, Rademaker AW, Pauloski BR, Kahrilas PJ (1993) Chin-down posture effect on aspiration in dysphagic patients. Arch Phys Med Rehabil 74:736–739

Sonies BC (1993) Remediation challenges in treating dysphagia post head/neck cancer. A problem-oriented approach. Clinics in communication disorders 3(4):21–26, fall

Splaingard ML, Hutchins B, Sulton LD, and Chauhuri G (1988) Aspiration in rehabilitation patients: videofluoroscopy vs. bedside clinical assessment. Arch Phys Med Rehabil 69: 637–640

Welch MV, Logemann JA, Rademaker AW, Kahrilas PJ (1993) Changes in pharyngeal dimensions effected by chin tuck. Arch Phys Med Rehabil 74:178–181.

Zenner PM, Losinski DS, Mills RH (1995) Using cervical auscultation in the clinical dysphagia examination in long-term care. Dysphagia 10(1): 27–31

5 Esophagus

Wolfgang Schima, Edith Eisenhuber, Arastoo Vossough, Marc S. Levine, Karin Aksglæde, Per Thommesen, Peter Funch-Jensen, David J. Ott, Benoit C. Pineau, Michael Y. Chen

5.1 Radiologic Evaluation of Esophageal Function

Wolfgang Schima and Edith Eisenhuber

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W. Schima, MD
Professor, Department of Radiology, University of Vienna, Währinger Gürtel 18–20, 1090 Vienna, Austria
E. Eisenhuber, MD
Department of Radiology, University of Vienna, Währinger Gürtel 18–20, 1090 Vienna, Austria

5.1.1 Introduction

Radiologic assessment of the esophagus is an essential part of the diagnostic work-up of patients with deglutition disorders. The radiologic examination comprises two parts: single- and double contrast examinations to assess the morphology of the esophagus and the esophago-gastric junction, which may reveal signs of esophagitis, tumour, strictures or rings. Radiologic evaluation of the esophagus would be incomplete without assessing esophageal function. Attempts to diagnose esophageal motor dysfunction, such as achalasia, were undertaken in the early days of single-contrast barium radiology (Hurst et al. 1930). The development of cinefluoroscopy and videofluoroscopy has significantly improved the ability to study the motor function of the pharynx and esophagus in detail. The pharyngeal and esophageal transport of liquid and solid boluses can be studied in real time and in slow motion. Although slow motion analysis is more crucial for the assessment of pharyngeal function, video recording of esophageal bolus transport is also essential for a thorough analysis of the esophagus. Subtle abnormalities of motor function may go undetected during real time observation of swallowing.

5.1.2 Normal Function of the Esophagus and the Lower Esophageal Sphincter

The esophagus is a tubular muscular structure, measuring approximately 23 cm in length (Li et al. 1994), which comprises outer longitudinal and inner circular muscle fibres. The proximal part of the esophagus consists of striated muscle fibres, whereas the distal part is composed of smooth muscle. The level of the transition zone between striated and smooth muscle is highly variable, with only the proximal 4 cm of the esophagus always composed of striated muscle. This dual structure of the esophageal musculature

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comprising striated muscle and smooth muscle fibres is significant in diseases that selectively affect either striated or smooth muscle. At the distal end of the esophagus, the tubular esophagus widens to the vestibular esophagus.

The lower esophageal sphincter between the vestibular esophagus and the stomach measures 3 to 5 cm in length. The lower esophageal sphincter corresponds to the high-pressure zone at the esophagogastric junction seen on esophageal manometry (Cohen 1979).

5.1.2.1 Primary and Secondary Peristalsis

Swallowing of a bolus triggers a primary peristaltic contraction wave in 95–96% of patients (Richter et al. 1987), which propagates with a velocity of 2–3.5 cm/s. Manometric studies have revealed that the lower esophageal sphincter has a resting pressure. Upon swallowing, the sphincter relaxes some seconds after triggering of swallowing. Radiologically, the lower esophageal sphincter is pushed open by the bolus arriving at the gastroesophageal junction (Dodds 1977). Immediately after bolus passage, the sphincter re-contracts (Dodds 1977). The amplitude and propagation velocity of primary peristalsis is modulated by bolus consistency, volume, and temperature (Dooley et al. 1988). If there is residual bolus in the esophagus or if there is a gastroesophageal reflux, a secondary peristaltic contraction wave can be triggered by the volume remaining in the esophagus to clear the esophagus. Both primary and secondary esophageal contractions are peristaltic and considered normal.

5.1.2.2 Nonpropulsive (or Tertiary) Contractions

Non-propulsive (or tertiary) contractions may also be seen in the esophagus during videofluoroscopy or manometry. They result in segmental muscular contractions, which are not propagated to the distal esophagus. They may occur simultaneously at multiple sites and they may be repetitive. They occur spontaneously or may be triggered by swallowing or other stimuli, such as acoustic emissions (Stacher et al. 1979). In young adults, these non-propulsive contractions are rarely seen upon swallowing (Richter et al. 1987). The prevalence and severity of non-propulsive contractions increases with age (Grishaw et al. 1996). Most often, they are signs of abnormal esophageal function.

5.1.2.3 Radiologic Evaluation of Esophageal Motor Function

Radiologic evaluation of esophageal function always includes assessment of the esophageal body as well as the pharyngo-esophageal and lower esophageal sphincters. In contrast to the assessment of pharyngeal function, esophageal peristalsis can be assessed in real-time. However, videotaping of a study is helpful to allow repeated analysis of bolus transport that would demonstrate subtle abnormalities. Esophageal function should be assessed in all patients who suffer from dysphagia or globus sensation when referred for a videofluoroscopic study. One must be careful in patients with clinically suspected aspiration. The study should always be initiated with an examination of pharyngeal function. If there is only laryngeal penetration or minimal aspiration of the contrast material, the examiner can proceed to assess esophageal function. In general, the administration of i.v. glucagon or Buscopan® should be avoided. Glucagon and Buscopan can alter the esophageal bolus transit and produce relaxation of the gastroesophageal sphincter, resulting in spontaneous gastroesophageal reflux (Anvari et al. 1989). For esophageal motor function studies, low density barium (app. 100% g/v) should be used. Barium at this consistency flows easily and is radio-opaque enough to provide good contrast in the esophagus. Since it is known that bolus viscosity alters peristalsis, thick high-density barium or barium paste should not be used. In case of aspiration, a limited study of esophageal function with iodinated, non-ionic contrast material may be considered.

For assessment of esophageal peristalsis, the observation of "single swallows" is essential. Repetitive swallowing inhibits the propagation of esophageal peristalsis (Meyer et al. 1981), a phenomenon, which is referred to as "deglutitive inhibition." If the patient swallows repeatedly within 5–10 sec, every new peristaltic contraction generated by swallowing will inhibit the preceding peristaltic contraction. Radiologically, this may be misinterpreted as impaired peristalsis or nonpropulsive contractions. After the end of a series of repeated swallows, a large contraction wave will "clear" the esophagus. Therefore, the size of the bolus administered is critical for assessment of esophageal peristalsis: we routinely use a bolus size of 10 ml of barium to ensure that the patient swallows only once. In contrast, rapid repetitive swallows maximally distend the esophagus for morphologic evaluation (e.g., the search for Schatzki rings).

Esophageal peristalsis is assessed in the upright and in the prone oblique position. Usually, swallows in the prone oblique position provide more information, because gravity does not support bolus transit. The peristaltic contraction wave occludes the esophageal lumen, giving the bolus tail typically an inverted "V"-shape (Fig. 5.1.1). It propels the complete bolus through the esophagus into the stomach. The proximal escape of a small amount of barium is not considered abnormal (Schima et al. 1992). Swallows in the upright position are sometimes of value, because they may reveal subtle motor abnormalities not seen in the prone oblique position (Sears et al. 1989). In the upright position, bolus transit through the esophagus is normally rapid. The persistence of an air-fluid level ("support level") is indicative of the presence of a disordered motor function or a stenosis (Schober et al. 1993).

The use of up to 10 swallows per patient during videofluoroscopy has been shown to be more sensitive for the detection of subtle motor abnormalities (Hewson et al. 1990). However, in clinical practice, the number of swallows observed must be limited because of radiation exposure, practicability, and patient comfort. Therefore, our routine examination protocol includes the observation of one bolus in the upright position and three boluses in the prone oblique position. The diagnostic value of videofluoroscopic studies in dysphagic patients can be increased by using solid barium-soaked marshmallows or globules, tablets, or rice (Aksglaede et al. 1992; Schwickert et al. 1993; Ott et al. 1991; Hannig et al. 1990).

5.1.3 Examination Technique (Schima et al. 1995)

1. Upright position, LPO (left posterior oblique): the patients takes a bolus of low-density barium and is asked to swallow only once. The LPO position is preferred to avoid superposition of the esophagus

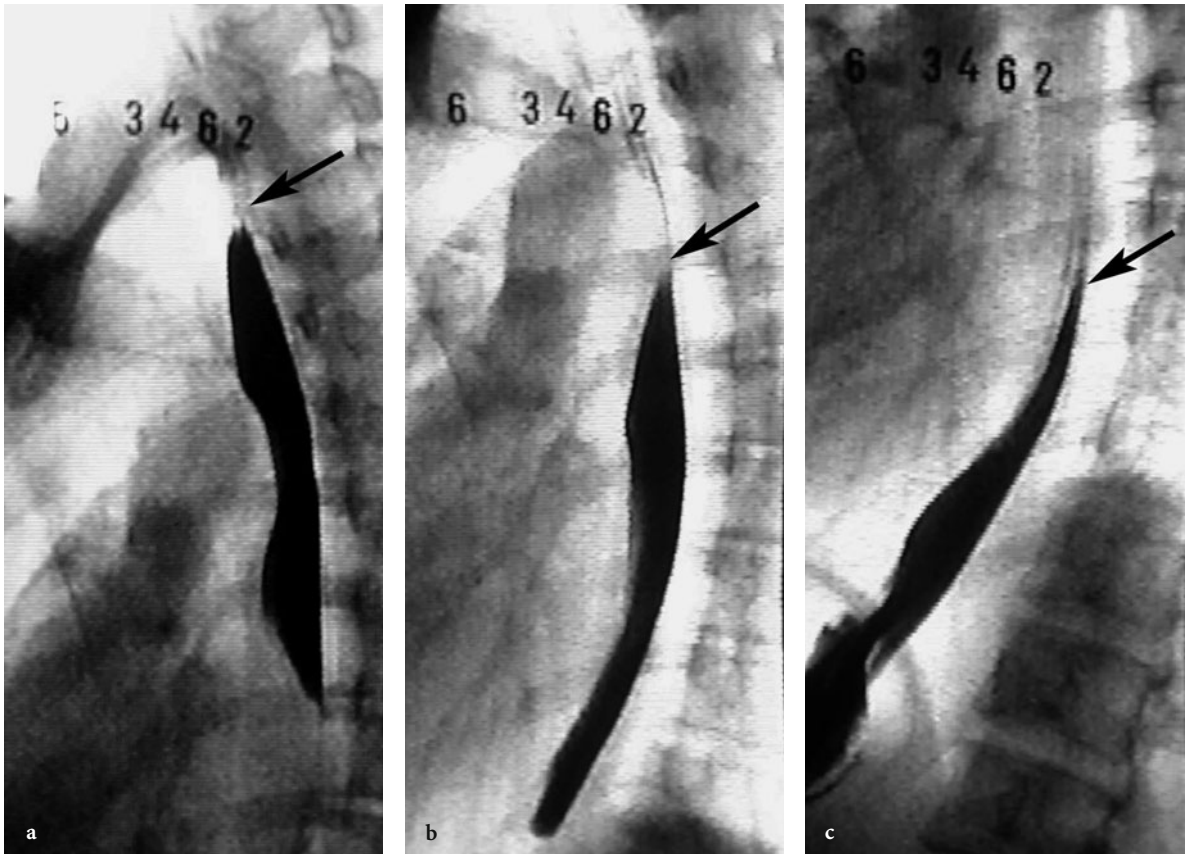

Fig. 5.1.1a-c. Normal esophageal motility. Patient is placed in the prone oblique position. There is a peristaltic contraction wave, which occludes the esophageal lumen, resulting in the typical inverted "V" shape (*arrows*) of the bolus tail. In Figure **a** to **c**, distal propagation of the bolus is evident

by the spine and to provide better visualization of the gastroesophageal segment.

2. After tilting of the table, the patient is turned to a prone oblique position. The patient takes a bolus with a straw. Up to three swallows are recorded. Imaging is centred on the tail of the bolus to look for proximal escape and stasis of the barium.
3. Assessment of the gastroesophageal reflux: reflux may or may not occur spontaneously during the examination. There are also provocative tests to elicit gastroesophageal reflux, including placing the patient in the Trendelenburg position, the water-siphon test, the Valsalva manoeuvre, and turning of the patient (Stewart 1981). We use only the latter two tests, which are more physiological than the former two. If reflux is noted during the examination, spot-film or video recording should be used to document it.
4. Solid bolus in the upright position: if the patient suffers from dysphagia for solids and the examination with liquid barium does not reveal the cause, a solid bolus (barium-soaked cookies, marsh mallows, tablets, etc.) may be used. Transit of solid food may be slow in some individuals. There is no standardized reference value for evaluation of solid food transit (Pouderoux et al. 1999). The best indicator for the presence of a significant stenosis (i.e., a Schatzki ring or a malignant stricture) or a motor abnormality is the induction of the typical symptoms of "food sticking in the throat" by a retained solid bolus.
5. Double-contrast and mucosal relief films of the esophagus and the lower esophageal sphincter (see Chapter on Esophageal Morphology)

Table 5.1.1. Classification of esophageal Motility Disorders

Primary Motility Disorders
Achalasia
Diffuse esophageal spasm
Nutcracker esophagus
Esophageal atresia
Non-specific esophageal motor disorders
Secondary Motility Disorders
Connective Tissue Diseases:
Progressive systemic sclerosis
Dermatomyositis
Polymyositis
Mixed connective tissue disease
Lupus erythematosus
Endocrine disease:
Diabetes mellitus
Myxoedema
Hyperthyroidism
Metabolic disorders:
Alcohol-induced
Amyloidosis
Infectious disorders:
Chagas disease
Candida
Herpes
Chemical:
Gastroesophageal reflux
Caustic agents
Muscular disorders:
Myasthenia gravis
Muscle dystrophy
Neurologic diseases:
Parkinson's disease
Guillain-Barré-syndrome
Poliomyelitis
Amyotrophic lateral sclerosis
Multiple sclerosis
Immunologic
Chronic graft-versus-host disease
Eosinophilic esophagitis
Iatrogenic
Medication
(anticholinergic agents, benzodiazepines, barbiturates, etc.)
Radiation
Post vagotomy

5.1.4 Esophageal Motility Disorders

Esophageal motor disorders can be divided into two major categories. Primary motor disorders occur independent of other diseases and include achalasia, diffuse esophageal spasm, non-specific esophageal motor abnormalities (NOMD), and nutcracker esophagus. Secondary motor disorders include a long list of motor abnormalities seen in conjunction with other diseases (Table 5.1.1). Classification of motor disorders is based on manometric findings. Diagnosis of a secondary motor abnormality requires, in addition, the diagnosis of an extra-esophageal disorder known to affect the esophagus. Esophageal motility disorders present with the non-specific symptom of dysphagia or chest pain. Although the clinical presentation may be the same in patients with different motor abnormalities, it is important to characterize the abnormality precisely. The optimal therapy is based on the specific knowledge of a manometric abnormality and may differ considerably between different groups. However, esophageal manometry is not widely available and in most cases not the first diagnostic test in patients with dysphagia. In these patients, either endoscopy or barium radiography is recommended in many institutions and countries (RCR Working Party 1998) (Czembirek et al. 2000). Radiographic assessment, in particular

videofluoroscopic recording, has been shown to be very useful in detecting and reliably characterizing esophageal motor abnormalities (Ott et al. 1987; Ott et al. 1990; Schima et al. 1992).

5.1.5 Primary Motor Disorders

5.1.5.1 Achalasia

Achalasia is the most widely known esophageal motor disorder. It is characterized by aperistalsis in the esophageal body and incomplete relaxation of the lower esophageal sphincter upon swallowing (Stacher et al. 1994; Richter 2001). The aetiology is not exactly known, but histopathologic lesions have been found in the dorsal motor nuclei of the brain stem, the vagal branches, and the myenteric plexus of the esophagus. Diagnosis of achalasia should be suspected when patients present with a long history of slowly progressive dysphagia for solids and liquids. Regurgitation of saliva and food immediately after swallowing (in contrast to gastroesophageal reflux) is common. Achalasia may also present as (non-cardiac) chest pain. Some patients complain of heartburn, despite the fact that incomplete opening of the lower esophageal sphincter is one of the key features of achalasia (Spechler et al. 1995). Esophageal manometry is the gold standard for the diagnosis of achalasia. Resting pressure of the lower esophageal sphincter is either normal or high, and there is incomplete relaxation of the sphincter upon swallowing. In the esophageal body, aperistalsis is present. Contractions are simultaneous and sometimes even of high amplitude (so-called "vigorous achalasia") (Goldenberg et al. 1991).

Radiologically, there is a typical appearance of esophageal dilatation with beak-like narrowing of the lower esophageal sphincter (Meshkinpour et al. 1992). Early in the disease, the esophagus has a normal diameter (Fig. 5.1.2). With progression of the disease, the esophagus becomes dilated and

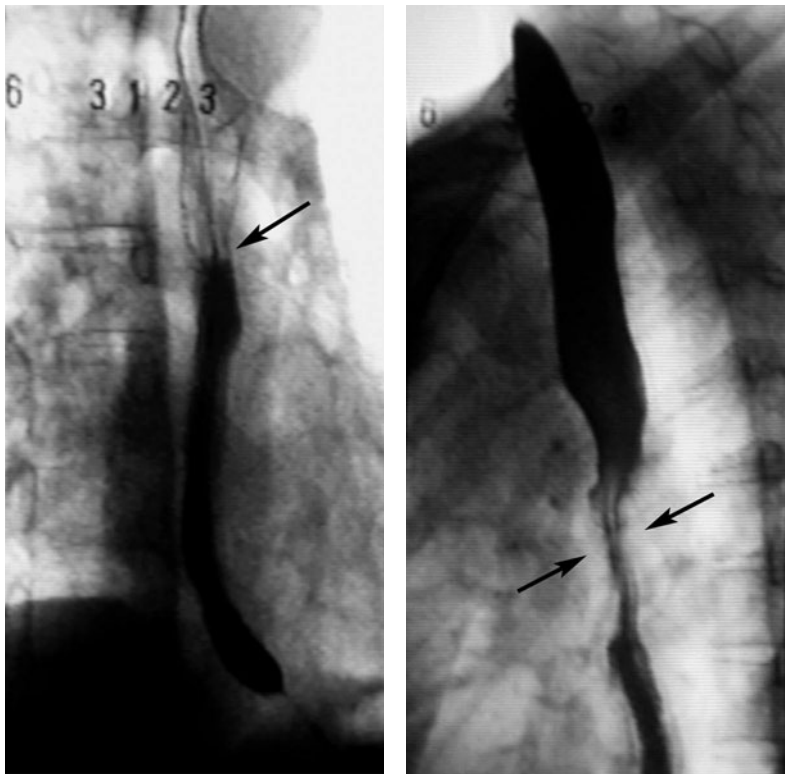

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Fig. 5.1.2a,b. Achalasia – early stage. **a** Videofluoroscopy in the upright position reveals a support level of contrast material indicative of delayed transit (*arrow*). The esophagus is not dilated. **b** In the prone oblique position there is aperistalsis. The arrow indicates a nonpropulsive contraction, which leaves most of the bolus retained in the proximal esophagus. Radiologic findings are indicative of a motor disorder, but diagnosis of a specific motor disorder can not be made. Subsequently, manometry revealed the presence of achalasia

retains food and saliva (Fig. 5.1.3). In advanced cases, esophageal dilatation may be severe (so-called "sigmoid esophagus") (Fig. 5.1.4) (Schima et al. 1993). Barium radiography has a low sensitivity in detecting achalasia, as alterations in esophageal morphology are present in advanced cases only. The radiologic staging system for achalasia according to Brombart is based on the grade of esophageal dilatation (< 4 cm, 4–6 cm, > 6 cm diameter) (Brombart 1980), which explains the low sensitivity of single-contrast upper gastrointestinal studies.

Multi-phasic radiographic evaluations including fluoroscopic assessment of esophageal motility without videotaping have been found to be more accurate in diagnosing achalasia. With videofluoroscopy, diagnosis is based not only on morphologic alterations of the esophagus, but also on assessment of functional abnormalities. Videofluoroscopy may reveal incomplete lower esophageal sphincter opening with delayed transit into the stomach. The feature of a transient support level of barium in the upright position is a non-specific sign, but this sign hints at the presence of either a motor abnormality or a distal stenosis (Fig. 5.1.2). The sensitivity of radiologic studies (either barium radiography or videofluoroscopy) for the detection of achalasia has been reported to be 58–95% (Howard et al. 1992; Ott et al. 1987; Schima et al. 1992; Schima et al. 1998). The wide range of reported sensitivities can be explained by variations in examination technique and patient populations. As described, the diagnosis is much more difficult to make in patients with early stages of the disease when esophageal dilatation is not yet present.

Several studies have reported a relation between achalasia and esophageal carcinoma. Patients with long-standing achalasia are at increased risk of carcinoma. The exact cause is unknown, but chronic stasis of food and saliva has been suggested. The reported incidences range from 1.7 to 20% (Meijssen et al. 1992). In a large prospective trial, the risk of developing cancer in patients with achalasia was found to be increased 33-fold, for a total of 3.4 cancers/1000 patients per year. Close follow-up of patients with achalasia is therefore strongly recommended (Meijssen et al. 1992).

5.1.5.2 Pseudoachalasia (Malignancy-induced Achalasia)

Malignancies involving the gastroesophageal junction can result in a clinical syndrome, pseudoachalasia, that mimics idiopathic achalasia. Pseudoachalasia is most often caused by adenocarcinoma of the

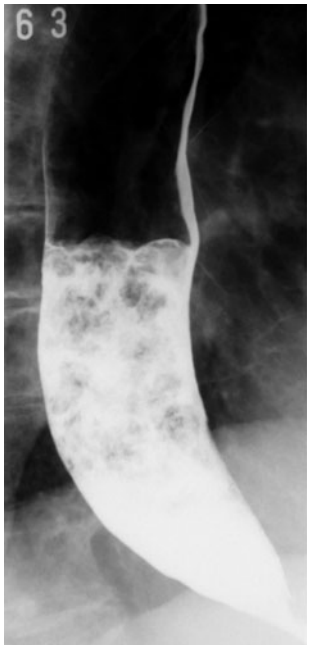

Fig. 5.1.3. Advanced achalasia. Barium radiography reveals moderate esophageal dilatation with retention of barium and secretions. There is the typical beak-like narrowing of the lower esophageal sphincter

Fig. 5.1.4. Long-standing achalasia. In this patient with a 36-year history of untreated achalasia, there is massive dilatation of the esophagus, which nearly fills the right hemithorax (Schima et al: Syncope after eating. New Engl J Med 1993; 328: 1572. © 1993 Massachusetts Medical Society. All rights reserved)

fundus invading the distal esophagus. Other causes are squamous carcinoma of the distal esophagus and hematogenous metastatic disease of the gastroesophageal junction with submucosal spreading (Dodds et al. 1986; Parkman et al. 1993; Kahrilas et al. 1987). Conventional esophageal manometry may not differentiate between idiopathic achalasia and pseudoachalasia. However, the correct diagnosis can be determined in most cases by observing clinical history and radiologic features. The mean duration of dysphagia is much shorter in patients with malignant pseudoachalasia than in idiopathic achalasia (1.9 months vs. 4.5 years) (Woodfield et al. 2000) and there is pronounced weight loss over time in malignancy-induced achalasia (Reynolds et al. 1989; Tremble 1959). Radiologically, the narrowed segment is longer in pseudoachalasia (4.4 cm vs. 1.9 cm) and reveals nodularity and abrupt proximal borders rather than a beak-like narrowing (Fig. 5.1.5) (Woodfield et al. 2000). The muscle-relaxing effect of amyl nitrite inhalation can be used to help make the correct diagnosis during barium radiography. After administration, there is relaxation of the lower esophageal sphincter with a subsequent opening of 2 mm or more in sphincter diameter (Dodds et al. 1986). In pseudoachalasia with tumour infiltration, the sphincter is unaffected by amyl nitrite. Computed tomography is also helpful in differentiating the two syndromes. Circumferential thickening of the lower esophageal sphincter of less than 10 mm is indicative of idiopathic achalasia, whereas pseudoachalasia patients have CT findings of marked or asymmetric wall thickening or a mass (Carter et al. 1997). When pseudoachalasia is suspected on the basis of clinical history and radiologic features, negative findings at endoscopy or biopsy should be viewed with caution (Tremble 1959). Repeated biopsies, endoscopic ultrasound, or even surgical exploration may finally lead to the diagnosis of malignancy-induced pseudoachalasia.

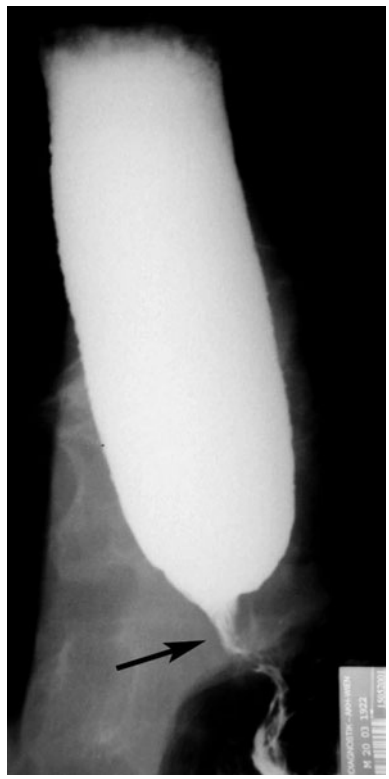

Fig. 5.1.5. Pseudoachalasia due to adenocarcinoma of the cardia. Barium radiography reveals moderate esophageal dilatation similar to that seen in achalasia. However, narrowing of the gastroesophageal junction does not appear beak-like. It is more irregular (*arrow*)

5.1.5.3 Diffuse Esophageal Spasm

The incidence of diffuse esophageal spasm is much lower than that of achalasia. Diffuse esophageal spasm is characterized by substernal chest pain, dysphagia, and the manometric evidence of simultaneous non-propulsive contractions on >10% of swallows with intermittent normal peristaltic contractions (Richter and Castell 1984). Differentiation between diffuse spasm and vigorous achalasia is based on the presence of normal relaxation of the lower sphincter in the former. However, diffuse esophageal spasm may evolve into vigorous achalasia or classic achalasia over time (Hannig and Wuttge-Hannig 1987).

The classic radiologic features of diffuse spasm are the presence of severe non-propulsive contractions causing esophageal curling or a "corkscrew" or "rosary bead" appearance (Chen et al. 1989) (Fig. 5.1.6). In two large studies by Ott et al., the correct diagnosis of diffuse spasm was made radiologically in 55–71% of patients (Ott et al. 1987; Ott et al. 1990). Incomplete or absent peristalsis and non-propulsive contractions are present in 71–76% of patients (Chen et al. 1989). However, radiologic findings are often non-specific and do not allow making the diagnosis of diffuse esophageal spasm. Therefore, patients with otherwise unexplained chest pain and radiologic evidence of a non-specific esophageal contraction abnormality should be referred for manometry.

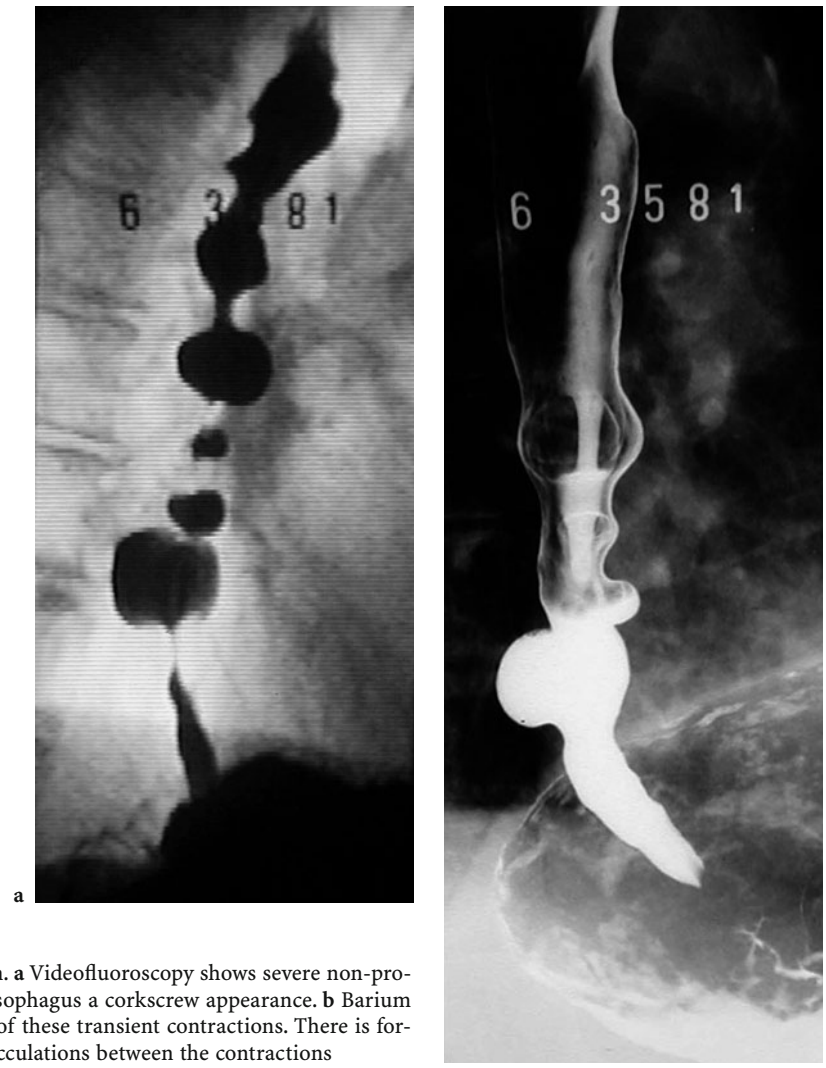

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Fig. 5.1.6a,b. Diffuse esophageal spasm. **a** Videofluoroscopy shows severe non-propulsive contractions, which give the esophagus a corkscrew appearance. **b** Barium radiography shows partial relaxation of these transient contractions. There is formation of pseudodiverticulum-like sacculations between the contractions

5.1.5.4
Nutcracker Esophagus

As more patients with non-cardiac chest pain were studied manometrically, an abnormality clearly different from diffuse spasm was recognized in 1979 (Benjamin et al. 1979). In the so-called "nutcracker esophagus," primary peristalsis is preserved, but there are peristaltic contractions of high amplitude and long duration. The diagnosis of nutcracker esophagus is made at manometry.

It is known that approximately 20% of patients admitted to cardiac care units show no abnormality in a detailed cardiac work-up (Bassotti et al. 1998). In a large percentage of these patients with non-cardiac chest pain, nutcracker esophagus or diffuse esophageal spasms are present.

Radiologically, the diagnosis is difficult, because peristalsis is intact. Chobanian et al. found non-specific abnormalities of esophageal bolus transit in 36% of patients (Chobanian et al. 1986). These findings were confirmed in a study by Ott et al.: in a series of 170 patients suffering from chest pain, nutcracker esophagus was even more prevalent than diffuse esophageal spasm, but a specific radiologic diagnosis could not be made in any of the patients (Ott et al. 1990).

5.1.5.5
Esophageal Atresia

In infants with esophageal atresia, Vogt's classification is based on the presence and location of an esophago-tracheal fistula (Hasse 1968). After esophageal repair, swallowing difficulties are common. The most

common source of postoperative dysphagia is the presence of strictures. However, esophageal dysfunction is also common (Auringer and Sumner 1994). There is absence of swallow-induced primary peristalsis and secondary peristaltic contractions have lower amplitudes than those seen in normal infants (Daum and Keuerleber 1969).

5.1.5.6 Non-specific Esophageal Motor Disorders (NOMD)

By far the most common esophageal motor disorders are non-specific contraction abnormalities. They may be idiopathic (primary) or secondary to a variety of extra-esophageal diseases (Table 5.1.1). Manometrically, contraction waves with multiple peaks, peristaltic waves with decreased amplitude, and isolated simultaneous or spontaneous contractions may be found (Gelfand and Botoman 1987). These contraction abnormalities do not fit into one of the above-mentioned categories of specific motor disorders.

Radiologically, incomplete or absent peristalsis and non-propulsive contractions can be seen. The sensitivity of radiographic studies is only 46–73%, because intermittent contraction abnormalities may elude radiographic detection (Ott et al. 1987; Schima et al. 1992). Clinically, it is important to search for underlying diseases, such as diabetes, alcoholism, or progressive systemic sclerosis, which may cause esophageal motility disorders (secondary motility disorders). In these cases, therapy is directed at the underlying disorder.

5.1.5.7 Presbyesophagus

In 1964, Soergel et al. reported a high incidence of esophageal motor abnormalities in elderly individuals, for which they coined the term "presbyesophagus" (Soergel et al. 1964). In their study on nonagenarians, non-propulsive contractions were prevalent in 10 of 15 patients. However, the existence of such a clinical entity has been much disputed. In another study (Hollis and Castell 1974), esophageal peristalsis was not found to be abnormal in elderly healthy individuals. It has been suggested that the increased prevalence of esophageal motor abnormalities is likely a function of an increased prevalence of underlying diseases, such as diabetes and neuromuscular disorders, which can affect esophageal motility (Ekberg and Feinberg 1991; Price and Castell 1978). It is most important in elderly individuals with newly developed dysphagia to rule out the presence of a tumour or a stricture before making the diagnosis of a motor disorder.

5.1.6 Secondary Motility Disorders

5.1.6.1 Progressive Systemic Sclerosis (PSS) and other Connective Tissue Diseases

Esophageal dysmotility is a well-known feature of progressive systemic sclerosis (or scleroderma) (Campbell and Schultz 1986) and other connective tissue diseases. PSS often affects the gastrointestinal tract, especially the esophagus and the small bowel, resulting in fibrosis and atrophy of smooth muscle. Esophageal involvement in PSS where the smooth muscle segment is affected results in hypomotility of the distal esophagus with absence of peristalsis and a patulous lower esophageal sphincter. Gastroesophageal reflux is common because of the incompetent sphincter, and refluxed acidic gastric contents are not readily cleared from the esophagus by secondary peristalsis. Esophageal symptoms, especially heartburn and dysphagia, are common in PSS. Such symptoms are found in up to 50% of patients (Sprung and Gibb 1985).

In the early stages of esophageal involvement, there is weak peristalsis in the distal esophagus (Montesi et al. 1991). Radiographically, the esophagus may be air-distended for a prolonged period after swallowing, without exhibiting the typical swallowing-induced collapse of the lumen due to a peristaltic contraction. In the prone oblique position, hypomotility is present in the distal esophagus with retention of barium (Fig. 5.1.7). With more advanced disease, esophageal dilatation and a patulous lower esophageal sphincter are apparent (Fig. 5.1.8). In the prone oblique position, complete aperistalsis with severe retention of barium (and saliva) will be found. Oropharyngeal dysfunction, including pharyngeal retention and aspiration, is found in 26% of patients (Montesi et al. 1991). Patients with an oropharyngeal disorder have a higher incidence of PSS-related pulmonary disease.

As esophageal dysmotility progresses, gastroesophageal reflux and its sequelae will predominate. Severe reflux esophagitis, strictures, and Barrett esophagus develops (Fig. 5.1.8). In the early advanced stages of the disease, differentiation between PSS and achalasia can be made with a high level of certainty. Although aperistalsis and esophageal dilatation is present in both diseases, the shape of the gastroesophageal junc-

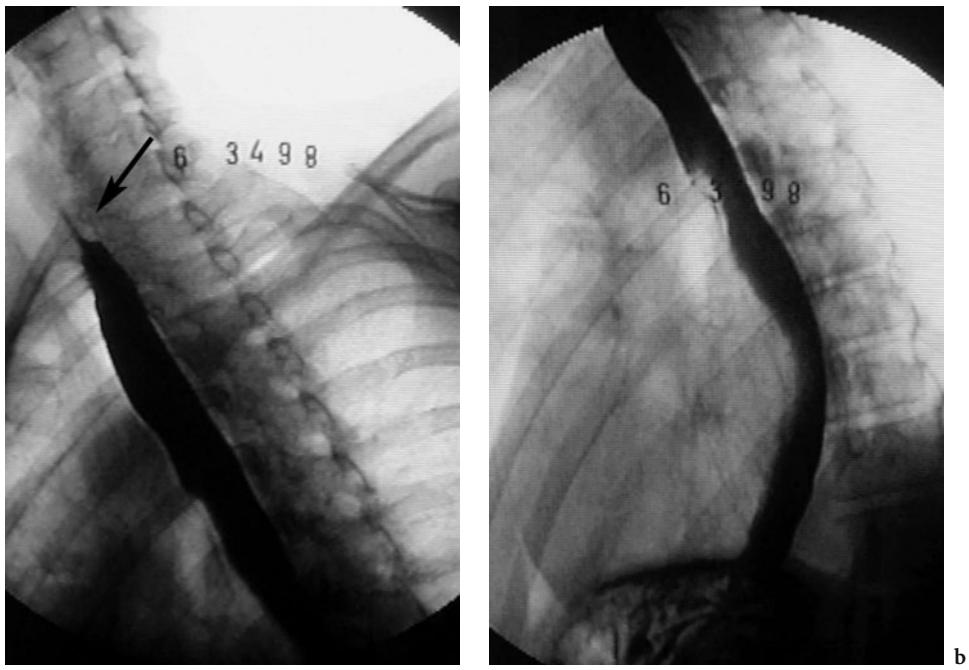

Fig. 5.1.7.a, b Progressive systemic sclerosis: early stage. **a** Videofluoroscopy in the prone oblique position shows normal peristaltic contraction in the proximal, striated muscle part of the esophagus. **b** The peristaltic wave subsides in the middle third of the esophagus with massive retention of barium, indicative of hypomotility. Together with clinical history, this is typical of esophageal involvement in PSS

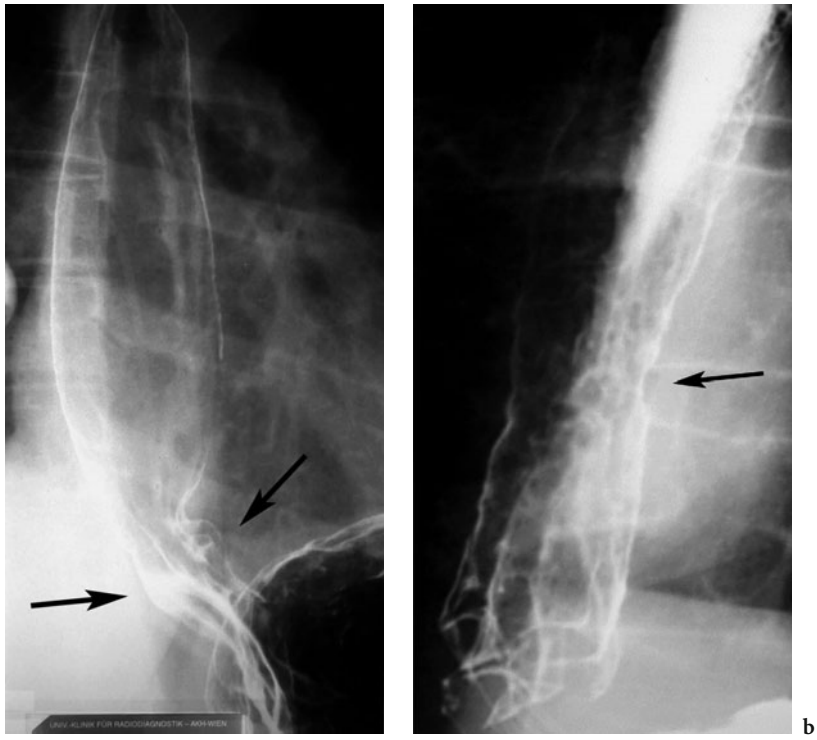

Fig. 5.1.8a,b. Progressive systemic sclerosis: advanced disease. **a** Barium radiography demonstrates esophageal dilatation and a widely patent gastroesophageal junction (*arrows*). **b** In another patient, there is obvious distal fold thickening indicative of reflux esophagitis. Barium radiography also shows a slight peptic stricture in the distal esophagus (*arrow*)

tion is markedly different. However, the development of distal peptic strictures in PSS may be confusing, although these strictures almost never have the bird beak-like appearance seen in achalasia.

PSS patients with severe reflux esophagitis are at increased risk of developing Barrett esophagus and, subsequently, adenocarcinoma (Sprung et al. 1985). Although barium radiography and videofluoroscopy are very sensitive (67–100%) for the detection of motor dysfunction in PSS (Campbell et al. 1986; Schima et al. 1992), these tests are not very accurate in the detection of peptic complications. For this reason, close endoscopic surveillance of PSS patients with reflux esophagitis and peptic strictures is recommended.

Esophageal dysmotility has also been reported to occur in patients with dermatomyositis/polymyositis and mixed connective tissue disease. Findings at radiography are non-specific, including low-amplitude peristalsis, aperistalsis, and delayed esophageal emptying at scintigraphy (Marshall et al. 1990; Horowitz et al. 1986).

5.1.6.2 Diabetes Mellitus

Esophageal symptoms are common in patients with diabetes, and the likelihood of dysphagia is more than three-fold higher compared to non-diabetic controls (Bytzer et al. 2001). Esophageal motor dysfunction has been demonstrated, characterized by weak peristalsis and increased frequency of non-propulsive contractions (Hollis et al. 1977; Holloway et al. 1999). Not surprisingly, a relation between the presence of esophageal dysmotility and diabetic neuropathy has also been reported (Mandelstam et al. 1969; Hollis et al. 1977). It has been suggested that autonomic neuropathy of the vagal nerve supplying the esophagus plays a major role in the development of diabetic dysmotility (Holloway et al. 1999). Radiographically, weak peristalsis or non-propulsive contractions can be observed (Borgström et al. 1988).

5.1.6.3 Chagas Disease

Chagas disease (South American trypanosomiasis) is caused by infection with the protozoon *Trypanosoma cruzi* (Dantas et al. 1999). In the chronic phase of the disease, it most often involves the heart, esophagus, and colon, causing cardiomegaly, megaesophagus, and megacolon. Chagas disease and achalasia share the same histopathologic lesion, and the loss of ganglion cells within the esophageal myenteric plexus (Dantas et al. 2001), which are destroyed by the infectious organism in Chagas disease. The clinical and radiological appearance of achalasia and Chagas disease may be identical (Fig. 5.1.9). Manometry may help to differentiate the two by identifying a higher resting pressure of the lower esophageal sphincter pressure in Chagas disease. The geographic origin of the patient may also provide a clue to the right diagnosis, and proof of Chagas disease is based on serologic testing.

5.1.6.4 Other Secondary Motility Disorders

There is a variety of other diseases and clinical conditions, that may affect the esophagus and cause a secondary motility disorder (Table 5.1.1), including amyloidosis (Rubinow et al. 1983; Lefkowitz et al. 1989; Burakoff et al. 1985), alcoholism (Grande et al. 1996), myxedema (Wright and Penner 1981), hyperthyroidism, Parkinsonism (Leopold and Kagel 1997), graft-versus-host disease (Schima

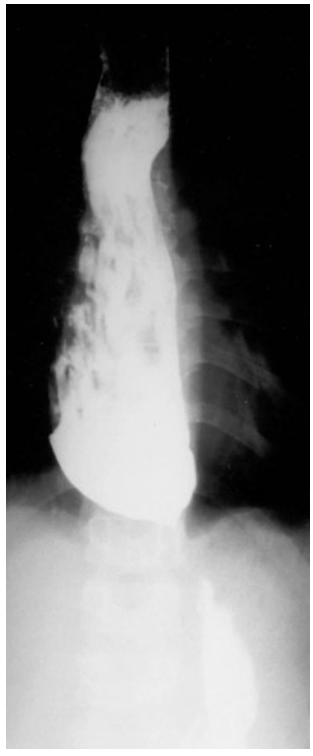

Fig. 5.1.9. Megaesophagus in a 14-year-old boy with Chagas disease. Barium radiography reveals esophageal dilation with tapering of the sphincter indistinguishable from idiopathic achalasia (*Image courtesy of Roberto Dantas, Ribeirão Preto, Brazil*)

et al. 1994), and Sjögren syndrome (Kjellén et al. 1986; Palma et al. 1994). In all these diseases, except progressive systemic sclerosis and Chagas disease, which give a typical radiographic appearance, non-specific esophageal function abnormalities have been found. Therefore, in all patients with an otherwise unexplained dysphagia and a non-specific esophageal contraction abnormality, the search should be directed towards the detection and therapy of an underlying disease.

5.1.7 Esophageal Diverticula Associated with Motility Disorders

Esophageal diverticula are included in this chapter because they are associated with an esophageal motility disorder in the vast majority of cases. Classification is based in the location: Zenker's diverticulum above the pharyngo-esophageal sphincter (i.e., a pharyngeal diverticulum that will not be covered in this chapter), midesophageal diverticulum just inferior to the level of the aortic arch, and epiphrenic diverticulum just above the diaphragm.

5.1.7.1 Midesophageal Diverticula

In the past, midesophageal diverticula have been widely considered to be traction-type diverticula of no clinical significance (Schmidt et al. 1991). Recently, this view has been questioned by some studies, which have shown that these diverticula resemble more the pulsion-type diverticula (Borrie and Wilson 1980; Evander et al. 1986). In 1974, Kaye reported on associated esophageal motor disorders found at manometry (Kaye 1974). In a series of 12 patients, diffuse spasms and nonspecific contraction abnormalities were the most common findings.

Based on radiographic findings, Rivkin et al. pointed out that midesophageal diverticula are likely of the pulsion-type. The pear-shaped configuration of most diverticula and their movement on swallowing resembles the appearance of Zenker's and epiphrenic diverticula (Rivkin et al. 1984). In our study including 30 patients with 33 midesophageal diverticula, 80% were diagnosed as propulsion-type diverticula based on radiographic findings. Diverticula were classified as pulsion-type when they were pear-shaped, when the size and shape changed during bolus passage, and when there was up- and downward movement of the diverticulum of at least 2 cm upon swallowing (Fig. 5.1.10) (Schima et al. 1997). In this study, 88% of patients with a pulsion diverticulum suffered from an esophageal motor disorder, and 6/20 (30%) cases were diagnosed as achalasia upon videofluoroscopy and manometry (Schima et al. 1997). In conclusion, midesophageal diverticula in symptomatic patients are primarily of the pulsion-type and tend to be associated with esophageal motor disorders.

5.1.7.2 Epiphrenic Diverticula

Epiphrenic diverticula are generally associated with and probably caused by an underlying esophageal motor dysfunction. Approximately two-thirds of patients will have specific motor disorders, with achalasia the most common (Bruggeman and Seaman 1973; Debas et al. 1980) (Fig. 5.1.11). The high percentage of motor abnormalities in association with epiphrenic diverticula determines the therapeutic and especially the surgical approach. Patients should always be referred for manometry to search for a curable manometric disorder before surgical resection of a "symptomatic" midesophageal diverticulum (Fig. 5.1.10). In these cases, diverticulectomy alone carries the risk of postoperative suture breakdown and predisposes the patient to later recurrence of the diverticulum (Rivkin et al. 1984). Myotomy of the lower esophageal sphincter is now a routine part of the operation (Evander et al. 1986).

5.1.8 Gastroesophageal Reflux Disease (GERD) and Esophageal Function

The term GERD covers the entire spectrum of clinical conditions and histologic esophageal alterations that result from gastroesophageal reflux (Dodds 1988). GERD is by far the most common cause of esophagitis in the general population. In the last 15 to 20 years, our knowledge about the aetiology and pathogenesis of gastroesophageal reflux and the development of esophagitis has considerably broadened. The pathogenesis is multifactorial and the factors believed to be important include (1) inadequate antireflux mechanisms, (2) chemical consistency of refluxed material, (3) esophageal clearance of refluxed material, (4) esophageal mucosal resistance, and (5) volume of gastric contents and efficacy of gastric emptying (Dodds et al. 1981; Dodds 1988).

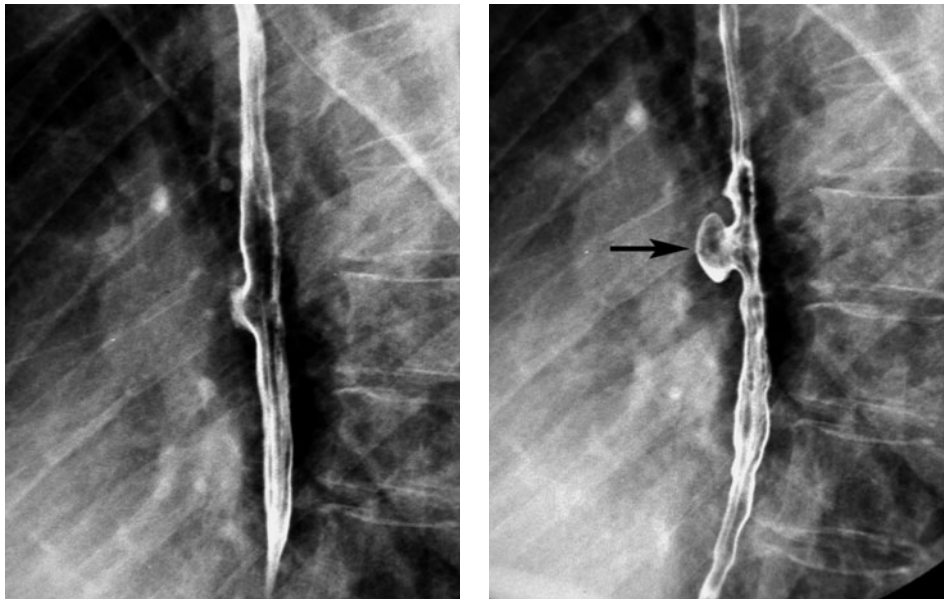

Fig. 5.1.10a,b. Midesophageal propulsion diverticulum in a patient with non-specific motor disorder. **a** Spot-film of the esophagus taken during deglutition is normal **b** Another spot-film taken approximately 2 sec later shows outpouching of a propulsion diverticulum in the mid-esophagus (arrow). A non-specific motor disorder was found at videofluoroscopy

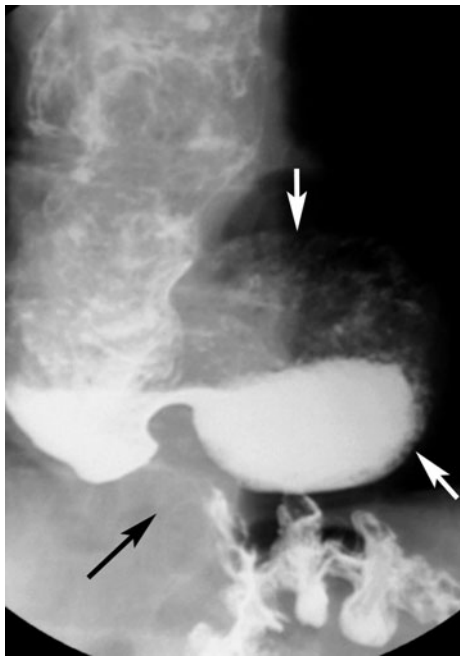

Fig. 5.1.11. Giant epiphrenic diverticulum in a patient with achalasia. Barium radiography demonstrates massive esophageal dilatation and a severe narrowing of the gastroesophageal junction, indicative of achalasia (*black arrow*). At videofluoroscopy, there was only a minimal transient opening of the sphincter with passage of only small amounts of barium. There is a giant epiphrenic diverticulum (*white arrows)* located above the sphincter

5.1.8.1 Hiatal Hernia and Reflux

In the past, the finding of a hiatal hernia has been considered the most important predisposing factor for the development of reflux. The exact prevalence of hiatal hernia in the general population remains unknown, largely because of the differences in examination techniques and diagnostic criteria (Fransson et al. 1989; Ott et al. 1985; Kahrilas et al. 1999). The relationship between the presence of a hiatal hernia and reflux disease remains controversial. Several studies have found that a hiatal hernia is much more common in patients with symptomatic reflux or abnormal findings at pH testing than in patients without reflux disease (prevalence, 80–94% vs. 59–60%) (Chen et al. 1992; Ott et al. 1985). Moreover, the presence of a hiatal hernia reduces lower esophageal sphincter pressure, which may increase the susceptibility to reflux events (Kahrilas et al. 1999). However, severe reflux esophagitis can also be found in patients without hiatal hernia (Kaul et al. 1986). Thus hiatal hernia is a non-specific radiologic finding with a poor predictive value: the presence of a hiatal hernia does not predict the presence of GERD (Ott et al. 1995). Conversely, the absence of a hiatal hernia does not exclude severe reflux esophagitis (Kaul et al. 1986).

5.1.8.2 Gastroesophageal Reflux and Esophageal Function

Effective esophageal peristalsis is a prerequisite for appropriate esophageal clearance of refluxed gastric contents. In normal subjects, a secondary peristaltic contraction is triggered by gastric contents refluxed into the esophagus, which rapidly clears the esophagus of the irritating agent. In patients with GERD, abnormalities of esophageal function are a common finding (Stein et al. 1990; Schoeman and Holloway 1995). Motor function deteriorates with increasing severity of mucosal injury (Fibbe et al. 2001). Swallow-induced primary peristalsis is impaired in GERD patients . It has also been shown that patients with reflux disease exhibit a defect in the triggering of secondary peristalsis (Schoeman and Holloway 1995). Instillation of water boluses or distension of the esophagus by air frequently fails to elicit a peristaltic contraction in reflux patients.

Interestingly, Timmer et al. demonstrated that impairment of esophageal peristalsis remained unchanged after healing of esophagitis (Howard et al. 1994; Timmer et al. 1994). Likewise, esophageal motility did not recover after fundoplication despite significant improvement in clinical symptoms and endoscopic signs of esophagitis (Fibbe et al. 2001). These results can be interpreted in two ways: first, deterioration of esophageal motility is irreversible in GERD; or, second, esophageal motility dysfunction is a preexisting factor in the pathogenesis of reflux disease (Timmer et al. 1994). The lack of improvement of esophageal motility with healing of esophagitis explains the high recurrence of esophagitis (50%) at two months after discontinuation of omeprazole therapy (Howard et al. 1994). These facts support the hypothesis that dysmotility is rather induced by irreversible inflammatory changes of the esophagus. However, a conclusive answer to this question will require a large prospective study of reflux patients with normal motility to determine whether motility deteriorates over time.

Both the barium swallow and radionuclide transit studies are useful in detecting motor disorders in GERD patients. Abnormalities seen in reflux disease include weak or even absent primary peristalsis and nonperistaltic contractions (Ott 1994). Clear-

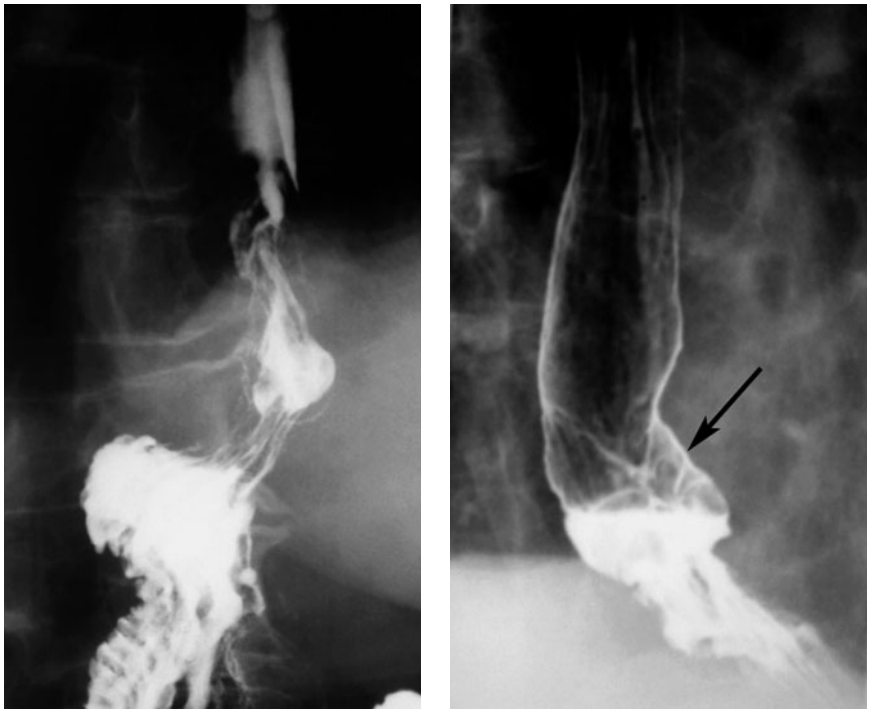

Fig. 5.1.12a,b. gastroesophageal reflux and reflux esophagitis. **a** In this patient with severe heartburn, there is spontaneous reflux of liquid barium and barium tablet in the right lateral position. **b** Double-contrast esophagram shows signs of severe reflux esophagitis in the distal esophagus with linear ulcerations (*arrow*)

ance of barium that has refluxed from the stomach can also be assessed in the recumbent position. At present, 24-h pH monitoring is the gold standard in the detection and quantitation of gastroesophageal reflux (Thompson et al. 1994). However, abnormal results at 24-h pH monitoring do not necessarily mean that the patient has clinical symptoms or endoscopic signs of esophagitis and vice versa. Results of several studies evaluating the role of radiology in patients with GERD have been disappointing (Chen et al. 1992; Kaul et al. 1986; Johnston et al. 1996). A major reason for the poor performance of radiology is that there is only limited time for fluoroscopic observation of barium. Meta-analysis of 9 studies on radiographic detection of gastroesophageal reflux revealed an average sensitivity of 39% (Ott 1994). In a study by Thompson et al., the diagnostic yield of spontaneous and provoked gastroesophageal reflux during barium radiography was assessed. The detection of spontaneous reflux revealed a sensitivity of 26% and a specificity of 94%. Using provocative tests, including cough/Valsalva manoeuvre, rolling, and the water-siphon test, increased the sensitivity of radiography to 31%, 44%, and 70%, respectively (Thompson et al. 1994). However, with increasing sensitivity the specificity dropped to 74%. Thus, prolonged observation and the use of provocative manoeuvres increases the sensitivity of barium radiography in the detection of reflux (Fig. 5.1.12). The absence of a reflux episode during fluoroscopy does not exclude the presence of GERD. Radiologic studies are not accurate enough to be used as a screening test in GERD patients: However, they may help to discover complications of reflux disease and to define the anatomy of the gastroesophageal junction in patients who are candidates for anti-reflux surgery.

5.1.9 Summary

In patients with dysphagia, radiographic studies evaluate both esophageal morphology and esophageal function. Videofluoroscopy accurately diagnoses achalasia, diffuse esophageal spasm, and progressive systemic sclerosis (scleroderma). Videofluoroscopy is less sensitive for the study of non-specific motor disorders and gastroesophageal reflux. When accuracy, costs, availability, and patient acceptance are considered (Parkman et al. 1996), videofluoroscopy is the initial diagnostic test for patients with esophageal dysphagia.

References

Aksglaede K, Funch-Jensen P, Vestergaard H, Thommesen P (1992) Diagnosis of esophageal motor disorders: a prospective study comparing barium swallow, food barium mixture, and continuous swallows with manometry. Gastrointest Radiol 17:1–4

Anvari M, Richards D, Dent J, Waterfall WE, Stevenson GW (1989) The effect of glucagon on esophageal peristalsis and clearance. Gastrointest Radiol 14:100–102

Auringer ST, Sumner E (1994) Pediatric upper gastrointestinal tract. Radiol Clin North Am 32:1051–1066

Bassotti G, Fiorella S, Germani U, Roselli P, Bataglia E, Morelli A (1998) The nutcracker esophagus: a late diagnostic yield notwithstanding chest pain and dysphagia. Dysphagia 13: 213–217

Benjamin SB, Gerhardt DC, Castell DO (1979) High amplitude, peristaltic esophageal contractions associated with chest pain and/or dysphagia. Gastroenterology 77:478–483

Borgström PS, Olsson R, Sundkvist G, Ekberg O (1988) Pharyngeal and esophageal function in patients with diabetes mellitus and swallowing complaints. Br J Radiol 61:817–821

Borrie J, Wilson RLK (1980) Esophageal diverticula: principles of management and appraisal of classification. Thorax 35: 759–767

Brombart MM (1980) Radiologie des Verdauungstraktes. Thieme, Stuttgart, New York, pp 244–245

Bruggeman LL, Seaman WB (1973) Epiphrenic diverticula: an analysis of 80 cases. AJR 119:266–276

Burakoff R, Rubinow A, Cohen AS (1985) Esophageal manometry in familial amyloid polyneuropathy. Am J Med 79:85–89

Bytzer P, Talley NJ, Leemon M, Young LJ, Jones MP, Horowitz M (2001) Prevalence of gastrointestinal symptoms associated with diabetes mellitus. Arch Intern Med 161:1989–1996

Campbell WL, Schultz JC (1986) Specificity and sensitivity of esophageal motor abnormality in systemic sclerosis (scleroderma) and related diseases: a cineradiographic study. Gastrointest Radiol 11:218–222

Carter M, Deckmann RC, Smith RC, Burrell MI, Traube M (1997) Differentiation of achalasia from pseudoachalasia by computed tomography. Am J Gastoenterol 92:624–628

Chen MYM, Ott DJ, Sinclair JW, Wu WC, Gelfand DW (1992) Gastroesophageal reflux disease: correlation of esophageal pH testing and radiographic findings. Radiology 185: 483–486

Chen YM, Ott DJ, Hewson EG, et al. (1989) Diffuse esophageal spasm: radiographic and manometric correlation. Radiology 170:807–810

Chobanian SJ, Curtis DJ, Benjamin SB, Cattau EL (1986) Radiology of the nutcracker esophagus. J Clin Gastroenterol 8: 230–232

Cohen S (1979) Motor disorders of the esophagus. N Engl J Med 301:184–192

Czembirek H, Frühwald F, Kainberger F (2000) Orientierungshilfe Radiologie. Anleitung zum optimalen Einsatz der klinischen Radiologie. 1st edn, ÖÄK Verlag, Wien, p 55

Dantas RO, Deghaide NHS, Donadi EA (1999) Esophageal manometic and radiologic findings in asymptomatic subjects with Chagas disease. J Clin Gastroenterol 28:245–248

Dantas RO, Deghaide NHS, Donadi EA (2001) Esophageal motility of patients with Chagas disease and idiopathic achalasia. Dig Dis Sci 46:1200–1206

Daum R, Keuerleber M (1969) Spätfunktion der intrathorakalen Speiseröhre nach operierter Ösophagusatresie. Kinderchir 7:49–60

Debas HT, Payne WS, Cameron AJ, Carlson HC (1980) Physiopathology of lower esophageal diverticulum and its implication for treatment. Surg Gynecol Obstet 151:593–600

Dodds WJ (1988) The pathogenesis of gastroesophageal reflux disease. AJR 151:49–56

Dodds WJ (1977) Current concepts of esophageal motor function: clinical implications for radiology. AJR 128:549–561

Dodds WJ, Hogan WJ, Helm JF, Dent J (1981) Pathogenesis of reflux esophagitis. Gastroenterology 81:376–394

Dodds WJ, Stewart ET, Kishk SM, Kahrilas PJ, Hogan WJ (1986) Radiologic amyl nitrite test for distinguishing pseudoachalasia from idiopathic achalasia. AJR 146:21–23

Dooley CP, Schlossmacher B, Valenzuela JE (1988) Effects of alterations in bolus viscosity of esophageal peristalsis in humans. Am J Physiol 254:G8–11

Ekberg O, Feinberg MJ (1991) Altered swallowing function in elderly patients without dysphagia: radiologic findings in 56 patients. AJR 156:1181–1184

Evander A, Little AG, Ferguson MK, Skinner DB (1986) Diverticula of the mid- and lower esophagus: pathogenesis and surgical management. World J Surg 10:820–828

Fibbe C, Layer P, Keller J, Strate U, Emmermann A, Zornig C (2001) Esophageal motility in reflux disease before and after fundoplication: a prospective, randomized, clinical and manometric study. Gastroenterology 121:5–14

Fransson S-G, Sökjer H, Johansson K-E, Tibbling L (1989) Radiologic diagnosis of gastro-esophageal reflux. Acta Radiologica 30:187–192

Gelfand MD, Botoman AV (1987) Esophageal motility disorders: a clinical overview. Am J Gastroenterol 82:181–187

Goldenberg SP, Burrell M, Fette GG, Vos C, Traube M (1991) Classic and vigorous achalasia: a comparison of manometric, radiographic, and clinical findings. Gastroenterology 101:743–748

Grande L, Monforte R, Ros E, et al. (1996) High amplitude contractions in the middle third of the esophagus: a manometric marker of chronic alcoholism? Gut 38:655–662

Grishaw EK, Ott DJ, Frederick MG, Gelfand DW, Chen MYM (1996) Functional abnormalities of the esophagus: a prospective analysis of radiographic findings relative to age and symptoms. AJR 167:719–723

Hannig C, Wuttge-Hannig A (1987) Röntgendiagnostik von Motilitätsstörungen des Pharynx und Ösophagus. Leber Magen Darm 1:7–17

Hannig C, Wuttge-Hannig A, Daschner H, Baum S, Güntner G (1990) Bariumsulfat–Gelatinekugeln zur Diagnostik spezieller pharyngoösophagealer Fragestellungen. Röntgenpraxis 43:15–19

Hasse W (1968) Ösophagusatresie. Thoraxchir 16:432–438

Hewson EG, Ott DJ, Dalton CB, Chen YM, Wu WC, Richter JE (1990) Manometry and radiology: complementary studies in the assessment of esophageal motility disorders. Gastroenterology 98:626–632

Hollis JB, Castell DO (1974) Esophageal function in elderly men: a new look at „presbyesophagus." Ann Intern Med 80: 371–374

Hollis JB, Castell DO, Braddom RL (1977) Esophageal function in diabetes mellitus and its relationship to peripheral neuropathy. Gastroenterology 73:1098–1102

Holloway RH, Tippett MD, Horowitz M, Maddox AF, Moten J, Russo A (1999) Relationship between esophageal motility and transit in patients with type I diabetes mellitus. Am J Gastroenterol 94:3150–3157

Horowitz M, McNeil JD, Maddern GJ, Collins PJ, Shearman DJC (1986) Abnormalities of gastric and esophageal emptying in polymyostitis and dermatomyositis. Gastroenterology 90:434–439

Howard JM, Reynolds RPE, Frei JV, et al. (1994) Macrosopic healing of esophagitis does not improve esophageal motility. Dig Dis Sci 39:648–654

Howard PJ, Maher L, Pryde A, Cameron EWJ, Heading RC (1992) Five years prospective study of the incidence, clinical features, and diagnosis of achalasia in Edinburgh. Gut 33:1011–1015

Hurst AF, Rake GW (1930) Achalasia of the cardia. Quart J Med 23:491–509

Johnston BT, Troshinsky MB, Castell JA, Castell DO (1996) Comparison of barium radiology with esophageal pH monitoring in the diagnosis of gastroesophageal reflux disease. Am J Gastroenterol 91:1181–1185

Kahrilas PJ, Kishk SM, Helm JF, Dodds WJ, Harig JM, Hogan WJ (1987) Comparison of achalasia and pseudoachalasia. Am J Med 82:439–446

Kahrilas PJ, Lin S, Chen J, Manka M (1999) The effect of hiatus hernia on gastro-esophageal junction pressure. Gut 44:476–482

Kaul B, Petersen H, Myrvold HE, Grette K, Røysland P, Halvorsen T (1986) Hiatus hernia in gastroesophageal reflux disease. Scand J Gastroenterol 21:31–34

Kaye MD (1974) Esophageal motor dysfunction in patients with diverticula of the mid-thoracic esophagus. Thorax 29:666–672

Kjellén G, Fransson SG, Lindström F, Sökjer H, Tibbling L (1986) Esophageal function, radiography, and dysphagia in Sjögren´s syndrome. Dig Dis Sci 31:225–229

Lefkowitz JR, Brand DL, Schuffler MD, Brugge WR (1989) Amyloidosis mimics achalasia's effect on lower esophageal sphincter. Dig Dis Sci 34:630–635

Leopold NA, Kagel MC (1997) Pharyngo-esophageal dysphagia in Parkinson's disease. Dysphagia 12:11–18

Li Q, Castell JA, Castell DO (1994) Manometric determination of esophageal length. Am J Gastroenterol 89:722–725

Mandelstam P, Siegel CI, Lieber A, Siegel M (1969) The swallowing disorder in patients with diabetic neuropathy-gastroenteropathy. Gastroenterology 56:1–12

Marshall JB, Kretschmar DC, Winship DC, Winn D, Treadwell EL, Sharp GC (1990) Gastrointestinal manifestations of mixed connective tissue disease. Gastroenterology 98:1232–1238

Meijssen MAC, Tilanus HW, van Blankenstein M, Hop WCJ, Ong GL (1992) Achalasia complicated by esophageal squamous cell carcinoma: a prospective study in 195 patients. Gut 33:155–158

Meshkinpour H, Kaye L, Elias A, Glick ME (1992) Manometric and radiologic correlations in achalasia. Am J Gastroenterol 87:1567–1570

Meyer GW, Gerhardt DC, Castell DO (1981) Human esophageal response to rapid swallowing: muscle refractory period or neural inhibition? Am J Physiol 241:G129–136

Montesi A, Pesaresi A, Cavalli ML, Ripa G, Candela M, Gabrielli A (1991) Oropharyngeal and eophageal function in scleroderma. Dysphagia 6:219–223

Ott DJ (1994) Gastroesophageal reflux disease. Radiol Clin North Am 32:1147–1166

Ott DJ, Gelfand DW, Chen YM, Wu WC, Munitz HA (1985) Predictive relationship of hiatal hernia to reflux esophagitis. Gastrointest Radiol 10:317–320
Ott DJ, Richter JE, Chen MY, Wu WC, Gelfand DW, Castell DO (1987) Esophageal radiography and manometry: correlation in 172 patients with dysphagia. AJR 149:307–311
Ott DJ, Abernethy WB, Chen MYM, Wu WC, Gelfand DW (1990) Radiologic evaluation of esophageal motility: results in 170 patients with chest pain. AJR 155:983–985
Ott DJ, Kelley TF, Chen MYM, et al. (1991) Evaluation of the esophagus with a marshmallow bolus: clarifying the cause of dysphagia. Gastrointest Radiol 16:1–4
Ott DJ, Glauser SJ, Ledbetter MS, Chen MYM, Koufman JA, Gelfand DW (1995) Association of hiatal hernia and gastroesophageal reflux: correlation between presence and size of hiatal hernia and 24-h pH monitoring of the esophagus. AJR 165:557–559
Palma R, Freire A, Freitas J, et al. (1994) Esophageal motility disorders in patients with Sjögren´s syndrome. Dig Dis Sci 39:758–761
Parkman HP, Cohen S (1993) Malignancy-induced secondary achalasia. Dysphagia 8:292–296
Parkman HP, Maurer AH, Caroline DF, Miller DL, Krevsky B, Fisher RS (1996) Optimal evaluation of patients with nonobstructive dysphagia. Manometry, scintigraphy, or videesophagography? Dig Dis Sci 41:1355–1368
Pouderoux P, Shi G, Tatum RP, Kahrilas PJ (1999) Esophageal solid bolus transit: studies using concurrent videofluoroscopy and manometry. Am J Gastroenterol 94:1458–1463
Price S, Castell DO (1978) Esophageal mythology. JAMA 240: 44–46
RCR Working Party (1998) Making the best use of a Department of Clinical Radiology: guidelines for doctors. 4th edn The Royal College of Radiologists, London, pp 50–52
Reynolds JC, Parkman HP (1989) Achalasia. Gastroenterol Clin North Am 18:223–255
Richter JE (2001) Esophageal motility disorders. Lancet 358: 823–828
Richter JE, Castell DO (1984) Diffuse esophageal spasm. Ann Intern Med 100:242–245
Richter JE, Wu WC, Johns DN, et al. (1987) Esophageal manometry in 95 healthy adult volunteers: variability of pressures with age and frequeny of „abnormal" contractions. Dig Dis Sci 32:583–592
Rivkin L, Bremner CG, Bremner CH (1984) Pathophysiology of mid-esophageal and epiphrenic diverticula of the esophagus. S Afr Med J 66:127–129
Rubinow A, Burakoff R, Cohen AS, Harris LD (1983) Esophageal manometry in systemic amyloidosis. Am J Med 75: 951–956
Schima W, Stacher P, Pokieser P, et al. (1992) Esophageal motor disorders: videofluoroscopic and manometric evaluation–prospective study in 88 symptomatic patients. Radiology 185:487–491
Schima W, Sterz F, Pokieser P (1993) Syncope after eating. N Engl J Med 328:1572
Schima W, Pokieser P, Forstinger C, et al. (1994) Videofluoroscopy of the pharynx and esophagus in chronic graft-versus-host disease. Abdom Imaging 19:191–194
Schima W, Pokieser P, Schober E (1995) Funktionsstörungen des Ösophagus: Radiologische Funktionsdiagnostik. Radiologe 35:693–702
Schima W, Schober E, Stacher G, et al. (1997) Association of midesophageal diverticula with esophageal motor disorders: videofluoroscopy and manometry. Acta Radiol 38: 108–114
Schima W, Ryan JM, Harisinghani M, et al. (1998) Radiographic detection of achalasia: diagnostic accuracy of videofluoroscopy. Clin Radiol 53:372–375
Schmidt R, Weidemann H, Bücherl ES (1991) Ösophagusdivertikel. Klinik und Therapie. Zentralbl Chir 116:89–93
Schober E, Schima W, Stacher G, Pokieser P, Uranitsch K, Tscholakoff D (1993) Yield of a sustained barium „support level" upon video-fluoroscopy in the diagnosis of esophageal motor disorders (abstract) 8th European Congress of Radiology, Scientific Programme and Abstracts, p 112
Schoeman MN, Holloway RH (1995) Integrity and characteristics of secondary esophageal peristalsis in patients with gastro-esophageal reflux disease. Gut 36:499–504
Schwickert HC, Schadmand-Fischer S, Klose P, Staritz M, Ueberschaer B, Thelen M (1993) Motilitätsstörungen des Ösophagus–Diagnostik mit dem Reisbreischluck. Fortschr Röntgenstr 159:511–517
Sears V, Castell J, Castell D (1989) „Abnormal" motility during upright and solid swallows in normal volunteers (abstract). Gastroenterology 96:A693
Soergel K, Zboralske FF, Amberg JR (1964) Presbyesophagus: esophageal motility in nonagenarians. J Clin Invest 43: 1472–1479
Spechler SJ, Souza RF, Rosenberg SJ, Ruben RA, Goyal RK (1995) Heartburn in patients with achalasia. Gut 37: 305–308
Sprung DJ, Gibb SP (1985) Dysplastic Barrett's esophagus in scleroderma. Am J Gastroenterol 80:518–522
Stacher G, Schmierer G, Landgraf M (1979) Tertiary esophageal contractions evoked by acoustic stimuli. Gastroenterology 77:49–54
Stacher G, Schima W, Bergmann H, et al. (1994) Sensitivity of radionuclide bolus transport and videofluoroscopic studies compared with manometry in the detection of achalasia. Am J Gastroenterol 89:1484–1488
Stein HJ, Eypasch EP, DeMeester TR, Smyrk TC, Attwood SEA (1990) Circadian esophageal motor function in patients with gastroesophageal reflux disease. Surgery 108:769–778
Stewart ET (1981) Radiographic evaluation of the esophagus and its motor disorders. Med Clin North Am 65: 1173–1194
Thompson JK, Koehler RE, Richter JE (1994) Detection of gastroesophageal reflux: value of barium studies compared with 24-h pH monitoring. AJR 162:621–626
Timmer R, Breumelhof R, Nadorp JHsM, Smout AJPM (1994) Esophageal motility and gastro-esophageal reflux before and after healing of reflux esophagitis. A study using 24-h ambulatory pH and pressure monitoring. Gut 35: 1519–1522
Tremble GE (1959) The clinical significance of a lump in the throat. Arch Otolaryngol 70:157–165
Woodfield CA, Levine MS, Rubesin SE, Langlotz CP, Laufer I (2000) Diagnosis of primary versus secondary achalasia: reassessment of clinical and radiographic criteria. AJR 175:727–731
Wright RA, Penner DB (1981) Myxedema and upper esophageal dysmotility. Dig Dis Sci 26:376–377

5.2 Esophageal Morphology

Arastoo Vossough and Marc S. Levine

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A. Vossough, MD; M. S. Levine, MD
Department of Radiology, Hospital of the University of Pennsylvania, 3400 Spruce Street, Philadelphia, PA 19104, USA

5.2.1. Inflammatory Conditions

5.2.1.1. Reflux Esophagitis

Reflux esophagitis is by far the most common inflammatory disease of the esophagus. The severity of reflux esophagitis depends not only on the frequency and duration of reflux episodes but also on the content of the refluxed material and the resistance of the esophageal mucosa (Pope 1994). Gastroesophageal reflux can be detected on barium studies, manometry, scintigraphy, and pH-monitoring techniques. However, reflux esophagitis can only be diagnosed by esophagography or endoscopy.

Reflux esophagitis may be manifested on single contrast barium studies by thickened folds, decreased distensibility, and marginal ulceration. However, these findings are detected only in patients with advanced disease. In contrast, double contrast barium studies have a sensitivity approaching 90% for the diagnosis of reflux esophagitis because of the ability to detect superficial ulcers or other findings that cannot be visualized on single contrast studies (Creteur et al. 1983b). Conversely, prone single contrast views permit optimal distention of the distal esophagus for demonstration of hernias, rings, or strictures that are sometimes missed on upright double contrast views (Chen et al. 1985).

Early reflux esophagitis may be manifested on double contrast studies by a finely nodular or granular appearance in the distal esophagus due to mucosal inflammation and edema (Fig. 5.2.1) (Kressel et al. 1981). In almost all cases, this nodularity or granularity extends proximally from the gastroesophageal junction as a continuous area of disease. As the disease progresses, other patients may develop shallow ulcers and erosions seen as streaks or dots of barium in the distal esophagus (Fig. 5.2.2) (Laufer 1982). These ulcers sometimes are associated with radiating folds or surrounding halos of edematous mucosa. Occasionally, the ulcers may be more widespread. However, ulceration in reflux esophagitis almost always involves

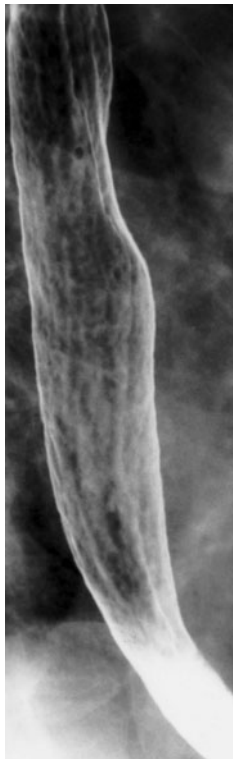

Fig. 5.2.1 Reflux esophagitis. Double-contrast view shows fine nodularity or granularity of mucosa in lower half of thoracic esophagus. Note how this granularity extends proximally from gastroesophageal junction as a continuous area of disease

the distal esophagus, so the presence of one or more ulcers that are confined to the upper or midesophagus should suggest another cause for the patient's esophagitis. Others with reflux esophagitis may have a single dominant ulcer, most commonly on the posterior wall of the distal esophagus (Hu et al. 1997). It has been hypothesized that these ulcers are located posteriorly because of prolonged exposure to refluxed acid that pools posteriorly when patients sleep in the supine position (Hu, Levine, and Laufer 1997).

Reflux esophagitis may also be manifested by thickened longitudinal folds due to inflammation and edema extending into the submucosa. These folds may have a smooth or lobulated contour, occasionally mimicking the appearance of esophageal varices. Other patients with chronic reflux esophagitis have a single prominent fold that arises in the gastric fundus and extends upwards into the distal esophagus as a smooth, polypoid protuberance, also known as an "inflammatory esophagogastric polyp-fold" (Fig. 5.2.3) (Bleshman et al. 1978). Because these lesions have no malignant potential, endoscopy is not warranted when typical esophagogastric polyps are found on double contrast studies.

Fig. 5.2.2 Reflux esophagitis. Double-contrast view shows superficial, punctate and linear ulcers (*arrows*) in distal esophagus just above hiatal hernia

Fig. 5.2.3 Inflammatory esophagogastric polyp. Single-contrast view shows a prominent fold (*straight arrow*) that arises at gastric cardia and terminates in distal esophagus as a smooth polypoid protuberance (*curved arrow*)

5.2.1.1.1. Peptic Scarring and Strictures

As esophageal ulcers heal, localized flattening or puckering of the esophageal wall may occur at the site of healing. Further scarring leads to the development of circumferential strictures (also known as "peptic" strictures) which typically appear as smooth, tapered areas of concentric narrowing in the distal esophagus above a hiatal hernia (Fig. 5.2.4). However, asymmetric scarring can lead to asymmetric narrowing with focal sacculation of the wall between areas of fibrosis. Scarring from reflux esophagitis can also lead to longitudinal shortening of the distal esophagus and the development of fixed transverse folds, producing a characteristic "stepladder" appearance due to pooling of barium between the folds (Fig. 5.2.5) (Levine and Goldstein 1984). These fixed transverse folds should be differentiated from the thin transverse folds ("feline" esophagus) often seen as a transient finding in patients with reflux.

5.2.1.1.2. Barrett's Esophagus

Barrett's esophagus is characterized by progressive columnar metaplasia of the distal esophagus due to long-standing acid reflux and reflux esophagitis. Although the metaplastic segment in Barrett's esophagus traditionally has been thought to extend 3 cm or more above the gastroesophageal junction, short-segment Barrett's esophagus has also been described (Yamamoto et al. 2001). Barrett's esophagus is important because it is associated with an increased risk of developing esophageal adenocarcinoma.

The classic radiologic sign of Barrett's esophagus is a high stricture or ulcer associated with a hiatal hernia and reflux (Fig. 5.2.6) (Levine 1995). However, strictures actually are more common in the distal esophagus in patients with this condition, so most cases do not fit the classic description of a high stricture or ulcer (Robbins et al. 1978). Another sign of Barrett's esophagus is a distinctive reticular pattern characterized by tiny barium-filled grooves resembling the areae gastricae on double contrast studies of the stomach (Fig. 5.2.7) (Levine et al. 1983). However, this reticular pattern is also found in only a small percentage of patients with Barrett's esophagus. Other more common findings in Barrett's esophagus, such as reflux esophagitis and peptic strictures, often occur in patients with uncomplicated reflux disease. Thus, radiographic findings that are relatively specific for Barrett's esophagus are not sensitive, and findings that are sensitive are not specific.

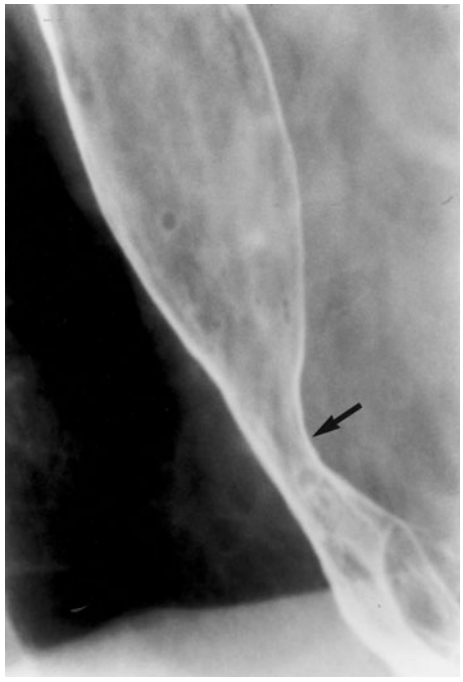

Fig. 5.2.4. Peptic stricture. Double-contrast view shows a smooth, tapered area of concentric narrowing (*arrow*) in distal esophagus above small hiatal hernia

Fig. 5.2.5 Mild peptic stricture with fixed transverse folds. Double-contrast view shows mild peptic stricture in distal esophagus with a series of incomplete transverse folds in region of stricture. Note how barium traps between folds (*arrows*), producing a characteristic "stepladder" appearance

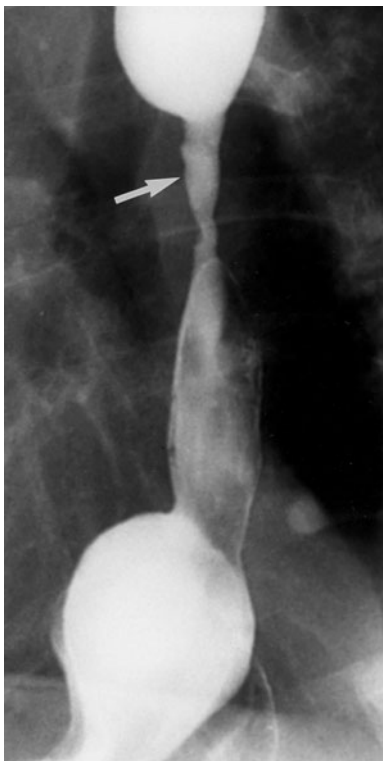

Fig. 5.2.6 Barrett's esophagus. Prone single-contrast view shows smooth, tapered stricture (*arrow*) in midesophagus above moderately large hiatal hernia

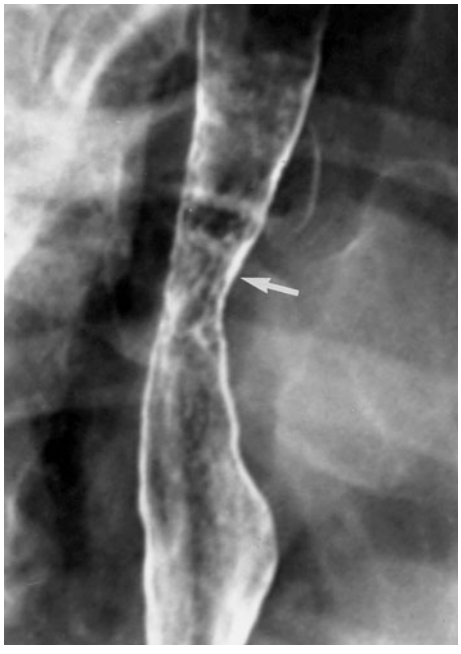

Fig. 5.2.7 Barrett's esophagus. Double-contrast view shows a tapered stricture (*arrow*) in midesophagus. Also note delicate reticular pattern of mucosa in region of stricture in this patient with proven Barrett's esophagus

Some investigators have employed a novel approach for evaluating patients with possible reflux disease. These patients were classified at high risk for Barrett's esophagus if double-contrast radiographs revealed a high stricture or ulcer or a reticular pattern; at moderate risk if the radiographs revealed a distal stricture or reflux esophagitis; and at low risk if the esophagus appeared normal (Gilchrist, Levine et al. 1988). The vast majority of those classified at high risk on double contrast barium studies were found to have Barrett's esophagus versus only 1% classified at low risk. Thus, the major value of double contrast esophagography in patients with possible reflux disease is its ability to stratify these individuals into various risk groups for Barrett's esophagus to determine the relative need for endoscopy and biopsy.

5.2.1.2. Infectious Esophagitis

5.2.1.2.1. Candida Esophagitis

Candida albicans is the most common cause of infectious esophagitis (Haulk and Sugar 1991). It most often occurs as an opportunistic infection in immunocompromised patients, particularly those with AIDS, but others may develop candidiasis as a result of local esophageal stasis due to achalasia or scleroderma (Gefter et al. 1981). It should be recognized that the absence of oropharyngeal candidiasis does not exclude the diagnosis of esophageal candidiasis (Levine et al. 1987). Single contrast barium studies have limited value in detecting this infection because of the superficial nature of the disease. In contrast, double contrast barium studies have a sensitivity of nearly 90% in diagnosing *Candida* esophagitis (Levine et al. 1985).

Candida esophagitis usually is manifested on double contrast studies by discrete plaque-like lesions corresponding to the characteristic white plaques seen on endoscopy (Levine et al. 1987). The plaques appear as linear or irregular filling defects which tend to be oriented longitudinally and are associated with normal intervening mucosa (Fig. 5.2.8). Patients with AIDS may develop a more severe form of *Candida* esophagitis characterized by a grossly irregular or "shaggy" esophagus due to coalescent plaques and pseudomembranes with trapping of barium between these lesions (Fig. 5.2.9) (Levine et al. 1987). Some of the plaques may eventually slough, producing deep ulcers on a background of diffuse plaque formation. Patients with scleroderma may also develop a "foamy" esophagus due to innumerable bubbles

Fig. 5.2.8 *Candida* esophagitis. Double-contrast view shows multiple discrete plaque-like lesions in midesophagus. Note how some plaques have a linear configuration. Also note how plaques are separated by segments of normal intervening mucosa

Fig. 5.2.9 Advanced *Candida* esophagitis with "shaggy" esophagus. Double-contrast view shows a grossly irregular esophagus due to innumerable coalescent plaques and pseudomembranes with trapping of barium between lesions. This patient had AIDS

layering out in the barium column (Fig. 5.2.10); this phenomenon presumably results from infection by the yeast form of candidiasis (Sam et al. 2000).

5.2.1.2.2. Herpes Esophagitis

The herpes simplex virus type I is the second most frequent cause of infectious esophagitis. Herpes esophagitis is most commonly seen in immunocompromised patients, but it occasionally may occur as an acute, self-limited disease in otherwise healthy patients who have no risk factors for opportunistic esophagitis (Shortsleeve and Levine 1992).

Herpes esophagitis initially is manifested by small vesicles that subsequently rupture to form discrete, punched-out ulcers on the mucosa (Levine et al. 1981). Affected individuals typically present with severe odynophagia. Although some patients may have herpetic lesions in the oropharynx, the majority are not found to have oropharyngeal disease. Moreover, some patients with oral herpes and odynophagia are found to have *Candida* esophagitis. The presence of other herpetic lesions therefore does not accurately predict herpes esophagitis in patients with odynophagia.

Herpes esophagitis usually is manifested on double contrast studies by small, discrete ulcers in the upper or midesophagus (Fig. 5.2.11). The ulcers can have a punctate, stellate, or volcano-like appearance, often separated by normal intervening mucosa (Levine et al. 1981). Discrete ulcers are seen in up to 50% of patients with herpes esophagitis (Levine et al. 1988). Rarely, severe infection may be manifested by multiple ulcers and plaques, mimicking the findings of advanced *Candida* esophagitis. In the appropriate setting, however, it usually is possible to differentiate these infections on the basis of the radiographic findings without need for endoscopy (Levine et al. 1987).

5.2.1.2.3. CMV Esophagitis

Cytomegalovirus (CMV) is another cause of infectious esophagitis associated with the development of ulcers, which can be single or multiple. Affected individuals usually present with severe odynophagia and are almost always found to have AIDS. These patients may also have evidence of CMV in other organs such as the retina, liver, or colon.

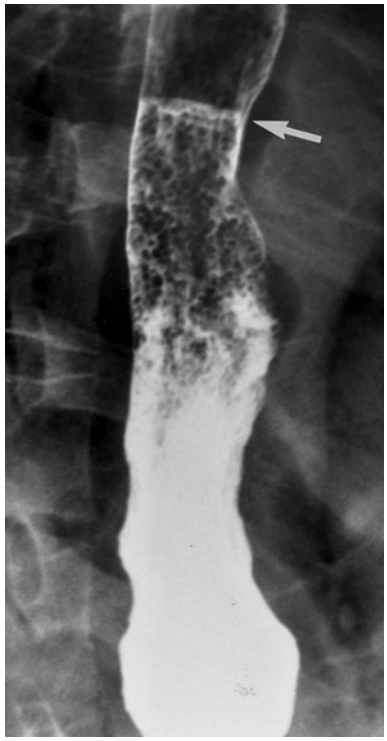

Fig. 5.2.10 *Candida* esophagitis with "foamy" esophagus. Double-contrast view shows innumerable tiny bubbles layering out in barium column (*arrow*) as a result of the yeast form of candidiasis. The esophagus also is dilated in this patient with underlying achalasia. It should be noted that patient did not receive effervescent agent for this examination

CMV esophagitis usually is manifested on double contrast barium studies by one or more giant, flat ulcers that are as large as several centimeters in length (Levine et al. 1987). These ulcers may have an ovoid, elongated, or diamond-shaped configuration and are frequently surrounded by a radiolucent rim of edematous mucosa. Because herpetic ulcers rarely become this large, the presence of one or more giant ulcers should suggest the possibility of CMV esophagitis in patients with AIDS. Nevertheless, the differential diagnosis also includes giant HIV ulcers in the esophagus (see next section). Less commonly, CMV esophagitis may be manifested by small, superficial ulcers indistinguishable from those of herpes esophagitis. CMV esophagitis is treated with potent antiviral agents such as ganciclovir which are associated with bone marrow toxicity. Endoscopy with biopsy specimens, brushings, and cultures from the esophagus therefore are required to confirm the presence of CMV before treating these patients.

5.2.1.2.4.
HIV Esophagitis

HIV infection of the esophagus can also lead to the development of giant ulcers which are indistinguishable from those caused by CMV (Levine et al. 1991b). The ulcers typically appear as ovoid or diamond-shaped collections, sometimes associated with a cluster of small satellite ulcers (Fig. 5.2.12). Affected indi-

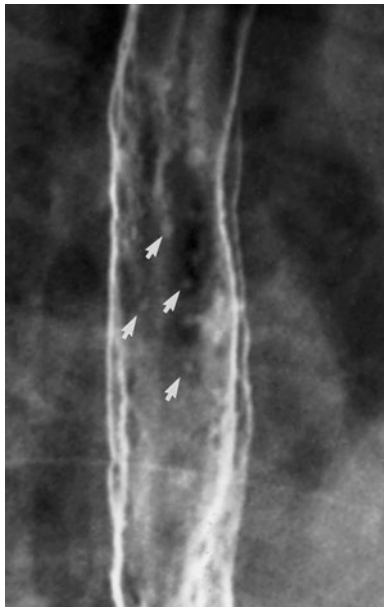

Fig. 5.2.11 Herpes esophagitis. Double-contrast view shows multiple tiny ulcers (*arrows*) in midesophagus. Note tiny radiolucent mounds of edema surrounding ulcers

Fig. 5.2.12 HIV esophagitis. Double-contrast view shows large diamond-shaped ulcer (*black arrows*) with a cluster of small satellite ulcers (*white arrows*) in midesophagus

viduals also can have palatal ulcers or a characteristic maculopapular rash on the upper half of the body. The diagnosis is confirmed by obtaining endoscopic biopsy specimens, brushings, and cultures from the esophagus to exclude the possibility of CMV. Unlike CMV ulcers, these HIV-related esophageal ulcers usually respond dramatically to treatment with oral steroids. Thus, endoscopy is required in HIV-positive patients with giant esophageal ulcers to differentiate HIV from CMV, so that appropriate therapy can be instituted (Sor et al. 1995).

5.2.1.3. Drug-Induced Esophagitis

Tetracycline and doxycycline are the most frequent causes of drug-induced esophagitis, but other offending agents include nonsteroidal anti-inflammatory drugs (NSAIDs), quinidine, potassium chloride, and alendronate. These patients typically ingest their medications with little or no water immediately before going to bed. It therefore has been postulated that prolonged contact of the esophageal mucosa with the medication causes an irritant contact esophagitis. Drug-induced esophagitis usually involves the upper or middle thirds of the esophagus with sparing of the distal third (Bova et al. 1987).

The radiographic findings in drug-induced esophagitis depend on the nature of the offending medication. Tetracycline and doxycycline are associated with the development of small, superficial ulcers indistinguishable from those of herpes esophagitis (Fig. 5.2.13). In contrast, quinidine, potassium chloride, and NSAIDs may cause more severe injury, leading to the development of large ulcers and subsequent stricture formation (Creteur et al. 1983a). Alendronate may also produce extensive ulceration and strictures, but these strictures are usually confined to the distal esophagus(Ryan et al. 1998). Whatever the offending agent, a repeat esophagram usually shows marked healing of the ulcers within 7-10 days after withdrawal of the offending medication (Creteur et al. 1983a).

5.2.1.4. Radiation Esophagitis

A radiation dose of approximately 4500 to 6000 rads to the chest can cause severe injury to the esophagus (Goldstein et al. 1975). Acute radiation esophagitis usually occurs 1-3 weeks after the initiation of radiation therapy. This condition may be manifested by ulceration and, even more commonly, by a granular appearance of the mucosa and decreased distensibility due to edema and inflammation of the irradiated segment (Collazzo et al. 1997). Within several months after completion of radiation therapy, a smooth, tapered area of concentric narrowing may

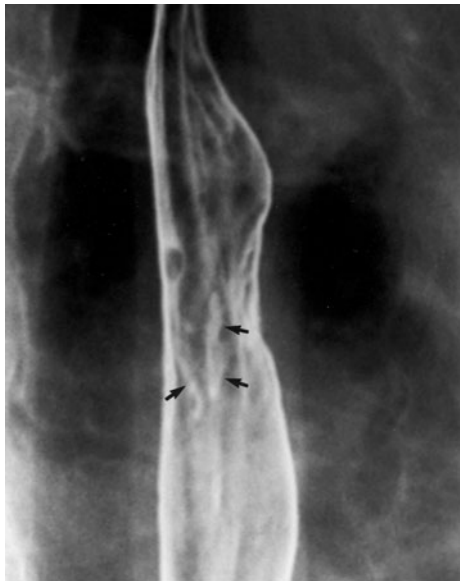

Fig. 5.2.13 Drug-induced esophagitis. Double-contrast view shows several small linear ulcers (*arrows*) in midesophagus due to recent tetracycline ingestion

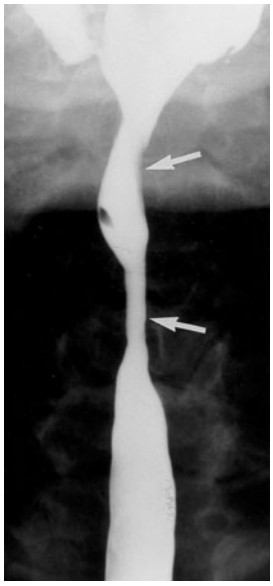

Fig. 5.2.14 Radiation stricture. A smooth, tapered stricture (*arrows*) is seen in cervical and upper thoracic esophagus in this patient who underwent total laryngectomy and radiation therapy for laryngeal carcinoma

be seen within the radiation portal due to the development of a radiation stricture (Fig. 5.2.14). Fistula formation is another uncommon complication of chronic radiation injury (Carlyle et al. 1976).

5.2.1.5. Caustic Esophagitis

Whether accidental or intentional, ingestion of lye or other caustic agents can lead to a severe form of esophagitis with ulceration, scarring, and stricture formation (Dafoe and Ross 1969). If esophagography is attempted after a patient ingests a caustic agent, water-soluble contrast media should be used because of the risk of perforation. Such studies may reveal abnormal motility, ulceration, or even esophageal disruption (Fig. 5.2.15). If the patient survives, repeat studies may reveal marked stricture formation, typically involving a long segment of the thoracic esophagus. Patients with chronic lye strictures also have an increased risk of developing squamous cell carcinoma of the esophagus, so a new area of mucosal irregularity within a pre-existing lye stricture should raise concern about the possibility of a superimposed carcinoma (Appelqvist and Salmo 1980).

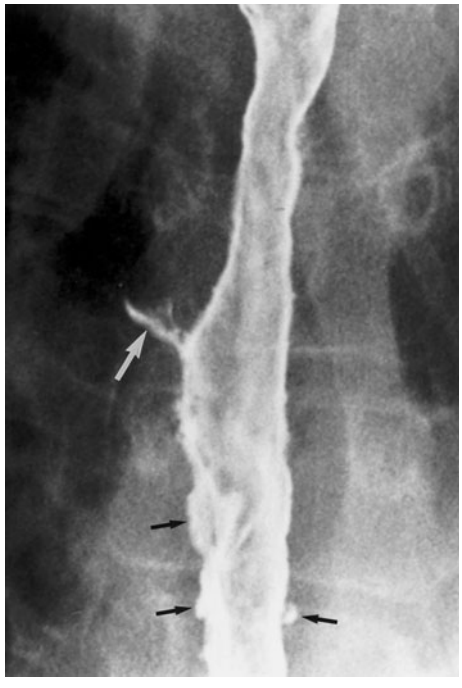

Fig. 5.2.15 Acute caustic esophagitis. Study with water-soluble contrast material shows narrowing and ulceration (*black arrows*) of midesophagus with small, sealed-off perforation (*white arrow*). This patient had ingested lye

5.2.1.6. Other Esophagitides

Alkaline reflux esophagitis is caused by reflux of bile or pancreatic secretions into the esophagus after partial or total gastrectomy (Levine et al. 1991a). These patients may develop mucosal nodularity, ulceration, or, in severe disease, rapidly progressive strictures in the distal esophagus (Levine et al. 1991a). The risk of alkaline reflux esophagitis can be decreased by performing a roux-en-Y esophagojejunostomy to prevent reflux of bile into the esophagus.

Nasogastric intubation is an unusual cause of esophagitis and stricture formation (Banfield and Hurwitz 1974). Most of these strictures develop after prolonged or repeated intubation, and they may progress rapidly after removal of the tube. It has been postulated that the strictures result from severe reflux esophagitis induced by the tube or from the irritant effect of the tube itself (Graham et al. 1959). Strictures caused by nasogastric intubation tend to be long-segment strictures in the distal esophagus which can progress rapidly on follow-up examinations.

Other less common causes of esophagitis include acute alcohol-induced esophagitis, Crohn's disease, eosinophilic esophagitis, chronic graft-versus-host disease, Behçet's disease, and, rarely, skin disorders involving the esophagus, such as epidermolysis bullosa dystrophica and benign mucous membrane pemphigoid.

5.2.2. Neoplasms

5.2.2.1. Benign Tumors

5.2.2.1.1. Papilloma

Squamous papillomas are the most common benign mucosal tumors in the esophagus. Histologically, these lesions consist of a central fibrovascular core with multiple digit-like projections covered by hyperplastic squamous epithelium (Miller et al. 1978). Papillomas usually appear on double contrast esophagography as small, sessile polyps with smooth or slightly lobulated borders. Because papillomas may be difficult to distinguish radiographically from early esophageal cancer and also because of the uncertain risk of malignant degeneration, biopsy or resection of esophageal papillomas is recommended by some investigators (Zeabart et al. 1979). Rarely, patients may have innumerable

papillomas in the esophagus, a condition known as esophageal papillomatosis (Sandvik et al. 1996).

5.2.2.1.2. Adenoma

Esophageal adenomas are rare benign lesions which almost always are found to arise in metaplastic columnar epithelium associated with Barrett's esophagus (Levine et al. 1984). Because these lesions have the same potential for malignant transformation as colonic adenomas, endoscopic or surgical resection is warranted (Levine et al. 1984). Adenomas typically appear on esophagography as sessile or pedunculated polyps in the distal esophagus near the gastroesophageal junction.

5.2.2.1.3. Glycogenic Acanthosis

Glycogenic acanthosis is a benign disorder in which glycogen accumulates in the squamous epithelial cell lining of the esophagus. It is a common degenerative condition, occurring primarily in middle-aged or elderly individuals (Glick et al. 1982). Glycogenic acanthosis is manifested on double contrast esophagography by multiple small, rounded nodules or plaques in the middle or, less commonly, distal esophagus (Glick et al. 1982). Major considerations in the differential diagnosis for these lesions include reflux and *Candida* esophagitis. However, the nodules in reflux esophagitis are more poorly defined and are usually located in the distal esophagus, whereas the plaques in Candida esophagitis tend to have a linear configuration and typically develop in immunocompromised patients with odynophagia. Thus, it usually is possible to differentiate these conditions on the basis of the clinical and radiographic findings.

5.2.2.1.4. Leiomyoma

Leiomyomas (gastrointestinal stromal tumors) are the most common benign submucosal tumors in the esophagus (Goldstein et al. 1981). Unlike stromal tumors elsewhere in the gastrointestinal tract, esophageal leiomyomas almost never undergo sarcomatous degeneration, and unlike gastric leiomyomas, they almost never ulcerate (Totten et al. 1953; Glanz and Grunebaum 1977). Patients with esophageal leiomyomas usually are asymptomatic but occasionally may present with slowly progressive dysphagia (Seremetis et al. 1976).

When esophageal leiomyomas grow into the mediastinum, they can be recognized on chest radiographs by the presence of a mediastinal mass, occasionally containing punctate areas of calcification. Leiomyomas usually appear on barium studies as smooth submucosal lesions (Fig. 5.2.16) indistinguishable from other mesenchymal neoplasms such as granular cell tumors, lipomas, hemangiomas, fibromas, and neurofibromas, except that leiomyomas are more likely on empirical grounds. CT may be helpful for differentiating submucosal esophageal masses from extrinsic mediastinal tumors compressing the esophagus.

5.2.2.1.5. Fibrovascular Polyp

Fibrovascular polyps are rare, benign submucosal tumors arising from the cervical esophagus, usually at the level of the cricopharyngeus (Levine et al. 1996). Histologically, these lesions are composed of fibrous, vascular, and adipose tissue covered by normal squamous epithelium. They gradually elongate into the thoracic esophagus, forming a pedunculated mass. Some fibrovascular polyps can grow to gigantic sizes, causing dysphagia or wheezing secondary to extrinsic compression of the trachea. Fibrovascular polyps usually appear on barium studies as smooth, expansile masses in the upper or midesophagus (Fig. 5.2.17), sometimes associated with a discrete pedicle that originates near the cricopharyngeus. When fibrovascular polyps contain a

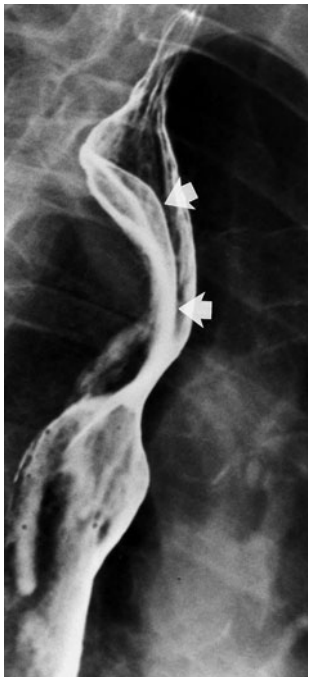

Fig. 5.2.16 Leiomyoma. Double-contrast view shows a smooth submucosal mass (*arrows*) in upper thoracic esophagus

Fig. 5.2.17 Giant fibrovascular polyp. A smooth, sausage-shaped mass is seen expanding lumen of upper and midesophagus on three separate views (*Black arrows denote tip of lesion in midesophagus*)

large amount of adipose tissue, they may be recognized by the presence of fat within the lesions on CT or MRI (Whitman and Borkowski 1989).

5.2.2.1.6. Granular Cell Tumor

Granular cell tumors arise from Schwann cells of the peripheral nervous system (Gershwind et al. 1978). Approximately 7% of granular cell tumors occur in the gastrointestinal tract, and one third of these lesions are found in the esophagus (Johnston and Helwig 1981). Granular cell tumors usually appear on esophagography as small, round or ovoid submucosal masses that often are mistaken for leiomyomas (Rubesin et al. 1985). Most patients with granular cell tumors are asymptomatic, so these lesions usually are detected as incidental findings. However, large granular tumors that cause dysphagia may require local excision (Rubesin et al. 1985).

5.2.2.1.7. Duplication Cyst

Although duplication cysts are not true neoplasms, they are included in this section as these lesions may present as submucosal masses. Esophageal duplication cysts comprise about 20% of all duplication cysts in the gastrointestinal tract (Macpherson 1993). These cysts are developmental anomalies of the primitive foregut, often containing ectopic gastric mucosa. The cysts may or may not communicate with the esophageal lumen and usually are located in the right lower mediastinum. Most adults are asymptomatic but some may have symptoms caused by obstruction, bleeding, or infection of the cyst.

Duplication cysts sometimes can be recognized on chest radiographs by the presence of a mass in the right lower mediastinum. The cysts typically appear on barium studies as smooth submucosal masses in the esophagus. When duplication cysts communicate with the esophageal lumen, they may be recognized as tubular, branching outpouchings that fill with barium (Fig. 5.2.18). Duplication cysts usually appear as homogenous low-attenuation structures on CT and as high-signal intensity structures on T2-weighted MR images (Rafal and Markisz 1991).

5.2.2.2. Malignant Tumors

5.2.2.2.1. Esophageal Carcinoma

Esophageal carcinoma constitutes about 1% of all cancers in the United States and 7% of all gastrointestinal

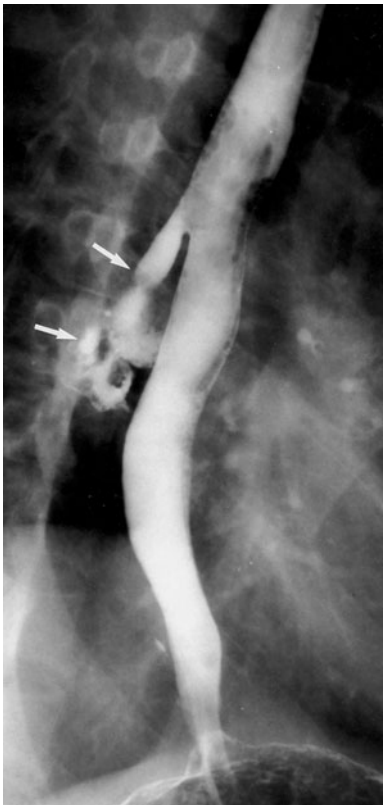

Fig. 5.2.18 Communicating duplication cyst. Single-contrast view shows a tubular, branching structure (*arrows*) that communicates with posterior wall of midesophagus (Courtesy of Marie Latour, M.D., Philadelphia, PA)

tumors (Livingston and Skinner 1985). Affected individuals usually develop dysphagia only after the tumor has invaded periesophageal lymphatics or other mediastinal structures. As a result, most patients have advanced lesions at the time of presentation, with overall 5-year survival rates of less than 10%. Occasionally, however, early esophageal cancer (defined as tumor limited to the mucosa or submucosa without lymphatic metastases) may be detected as a result of early onset of symptoms or screening of asymptomatic patients in high-risk groups. Unlike advanced carcinoma, early esophageal cancer is a curable lesion with 5-year survival rates as high as 95% (Levine et al. 1986). Histologically, 50–70% of esophageal cancers are squamous cell carcinomas and the remaining 30–50% are adenocarcinomas (Pera et al. 1993).

Tobacco and alcohol are the major risk factors for developing squamous carcinoma of the esophagus in the United States. Other conditions known to predispose to the development of esophageal carcinoma include achalasia, lye strictures, head and neck tumors, Plummer-Vinson syndrome, and tylosis (Carter and Brewer 1975; Appelqvist and Salmo 1980; Levine and Halvorsen 2000). Some authorities advocated periodic screening of patients with these conditions to detect developing cancers at the earliest possible stage.

Unlike squamous cell carcinomas, esophageal adenocarcinomas are virtually always found to arise in patients with underlying Barrett's esophagus. The reported prevalence of adenocarcinoma in patients with Barrett's esophagus is about 10% (Levine et al. 1995). Studies using incidence rather than prevalence data indicate that the relative risk of adenocarcinoma developing in patients with Barrett's esophagus is 30 to 40 times greater than that in the general population (Spechler et al. 1984). These adenocarcinomas evolve through a sequence of progressive epithelial dysplasia in areas of pre-existing columnar metaplasia. Some authorities therefore advocate periodic endoscopic surveillance of patients with known Barrett's esophagus in order to detect dysplastic or carcinomatous changes at the earliest possible stage.

Double-contrast esophagography has a sensitivity of more than 95% in detecting esophageal cancer, a figure comparable to the reported endoscopic sensitivity of 95-100% when multiple brushings and biopsy specimens are obtained (Levine et al. 1997). Furthermore, endoscopy rarely finds cases of esophageal carcinoma that are missed on double-contrast studies (DiPalma et al. 1984). We therefore believe that double-contrast esophagography is a sensitive technique for detecting esophageal cancer and that endoscopy is not warranted in patients with normal findings on double-contrast studies.

Early esophageal cancers usually are small, protruded lesions less than 3.5 cm in size. These tumors may be manifested on double-contrast studies by plaque-like lesions (often containing a flat central ulcer) (Fig. 5.2.19), by sessile polyps with a smooth or slightly lobulated contour, or by focal irregularity of the wall. Early adenocarcinomas in Barrett's esophagus may also be manifested by a localized area of wall flattening or irregularity within a pre-existing peptic stricture. Superficial spreading carcinoma is another form of early esophageal cancer characterized by a confluent area of nodules or plaques (Levine et al. 1986). Although these lesions may be confused with focal *Candida* esophagitis, the plaques in candidiasis tend to be discrete lesions with normal intervening mucosa, whereas the nodules in superficial spreading carcinoma tend to coalesce, producing a continuous area of disease. Although early esophageal cancers are generally thought to be small lesions, some early cancers may be relatively large intraluminal masses more than 3.5 cm in diameter (Levine et al. 1986).

Fig. 5.2.19 Early esophageal carcinoma. Double-contrast view shows a plaque-like lesion in midesophagus (*black arrows*) with flat, central ulcer (*white arrows*)

Advanced esophageal carcinomas usually appear on barium studies as infiltrating, polypoid, ulcerative, or varicoid lesions. Infiltrating carcinomas are manifested by irregular luminal narrowing, nodularity, ulceration, and abrupt, often shelf-like borders (Fig. 5.2.20). Polypoid carcinomas appear as lobulated intraluminal masses. Primary ulcerative carcinomas are necrotic lesions with a giant, meniscoid ulcer surrounded by a radiolucent rim of tumor (Fig. 5.2.21) (Gloyna et al. 1977). Finally, varicoid carcinomas are those in which submucosal spread of tumor produces thickened, tortuous longitudinal defects, mimicking the appearance of varices. However, varicoid tumors have a fixed configuration and relatively abrupt borders, whereas varices tend to change in size and shape at fluoroscopy. Also, varices rarely cause dysphagia because they are soft and compressible. Thus, it usually is possible to differentiate varices from varicoid tumors on clinical and radiologic criteria.

Squamous cell carcinomas and adenocarcinomas of the esophagus cannot be reliably differentiated on the basis of the radiographic findings. Nevertheless, squamous cell carcinomas tend to involve the upper or midesophagus, whereas adenocarcinomas are mainly located in the distal esophagus. Unlike squamous

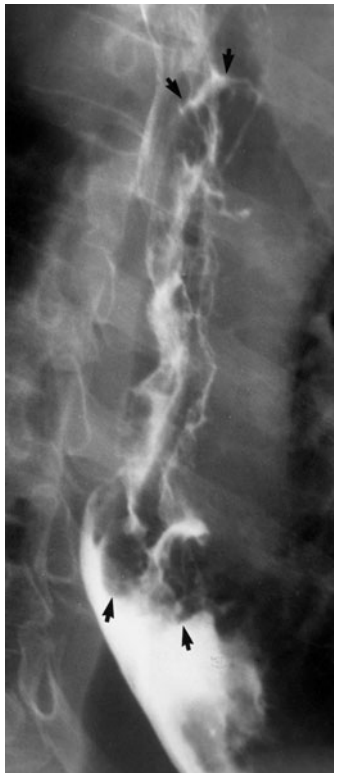

Fig. 5.2.20 Advanced infiltrating carcinoma. Single-contrast view shows an irregular segment of narrowing in midesophagus with abrupt, shelf-like proximal and distal borders (*arrows*)

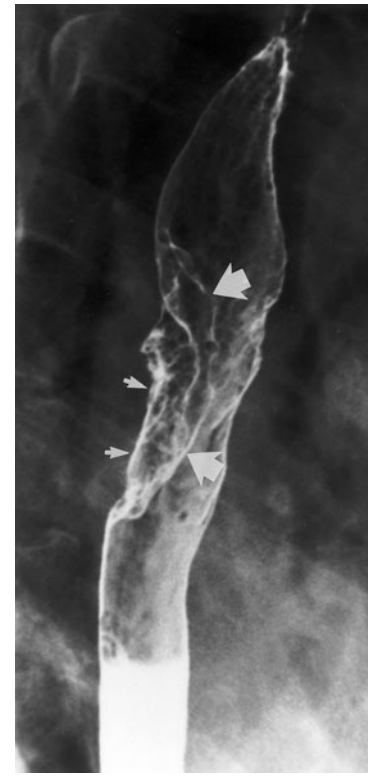

Fig. 5.2.21 Primary ulcerative carcinoma. Double-contrast view shows a polypoid lesion (*large arrows*) in midesophagus with flat area of central ulceration (*small arrows*)

carcinomas, esophageal adenocarcinomas also have a marked tendency to invade the gastric cardia or fundus. In fact, 50% of all cancers involving the gastroesophageal junction are esophageal adenocarcinomas invading the stomach.

Esophageal carcinomas tend to metastasize to other parts of the esophagus via a network of rich submucosal lymphatic channels. These lymphatic metastases may appear as polypoid, plaque-like, or ulcerated lesions that are separated from the primary tumor by normal intervening mucosa (Glick et al. 1986). Tumor may also spread subdiaphragmatically to the gastric fundus via submucosal esophageal lymphatic vessels. These metastases to the stomach may appear as large submucosal masses, often containing central areas of ulceration.

Appropriate treatment strategies for esophageal carcinoma depend on accurate staging of the tumor. Various imaging techniques such as CT, MR, and endoscopic sonography are used for staging esophageal carcinoma (Takashima et al. 1991; Vilgrain et al. 1990). The tumor stage is assessed by evaluating the depth of esophageal wall invasion and the presence or absence of lymphatic or distant metastases.

5.2.2.2.2. Other Malignant Tumors

Non-Hodgkin's lymphoma and, rarely, Hodgkin's lymphoma may also involve the esophagus. Esophageal lymphoma may be manifested on barium studies by submucosal masses, polypoid lesions, enlarged folds, or strictures (Carnovale et al. 1977). Spindle cell carcinoma is another rare tumor characterized by a polypoid intraluminal mass that expands the lumen of the esophagus without causing obstruction (Agha and Keren 1985). Other malignant tumors of the esophagus include leiomyosarcoma, malignant melanoma, and Kaposi's sarcoma.

5.2.3. Webs

Webs are thin mucosal folds arising from the anterior wall of the lower hypopharynx or upper cervical esophagus. Cervical esophageal webs have been reported in 3-8% of upper gastrointestinal barium studies (Ekberg and Nylander 1983). Most patients are asymptomatic, but some may present with dysphagia. An association of cervical esophageal webs with iron-deficiency anemia and pharyngeal carcinoma has been reported in northern Europe (also known as the Plummer-Vinson or Paterson-Kelly syndrome), but this association has not been observed in the United States (Ekberg and Nylander 1983).

Webs are characterized by 12 mm wide, shelf-like filling defects along the anterior wall of the hypopharynx or cervical esophagus that protrude to various depths into the esophageal lumen (Fig. 5.2.22). The use of dynamic or rapid sequence imaging and large barium boluses increases the detection rate of webs. In more severe cases, webs may be associated with a jet phenomenon and proximal dilatation of the pharynx (Taylor et al. 1990).

5.2.4. Rings

Lower esophageal rings are found in 6–14% of patients who undergo esophagography, but fewer than 1% of patients with rings are symptomatic (Schatzki and Gary 1956). The term Schatzki ring should be reserved only for those patients with lower esophageal rings who present with dysphagia. These rings almost always occur at the gastroesophageal junction. Histologically, the superior surface of the

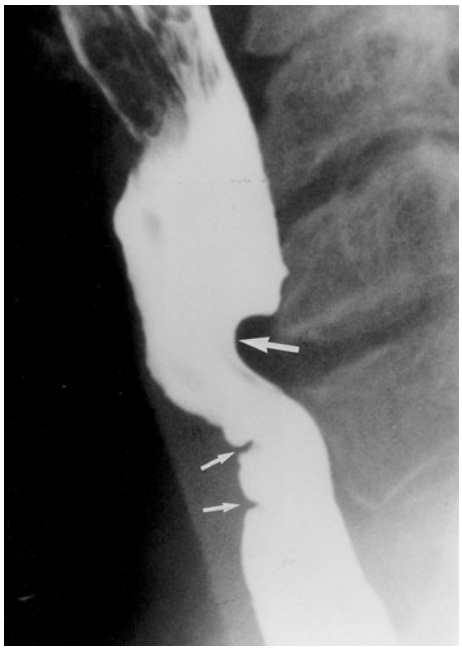

Fig. 5.2.22 Cervical esophageal webs. Lateral view of pharynx and cervical esophagus during swallowing shows two thin, web-like indentations (*small arrows*) on anterior wall of cervical esophagus. Also note rounded indentation posteriorly (*large arrow*) at pharyngoesophageal junction due to incomplete relaxation of cricopharyngeus

ring is lined by stratified squamous epithelium and the inferior surface by columnar epithelium. The exact pathogenesis of Schatzki rings is uncertain but some are thought to be develop as a result of scarring from reflux esophagitis (Chen et al. 1987).

Lower esophageal rings appear on barium studies as 2–3 mm in height, web-like constrictions at the gastroesophageal junction, almost always above a hiatal hernia (Fig. 5.2.23). The rings can be missed if the distal esophagus is not adequately distended at fluoroscopy, so it is important to obtain prone views of the esophagus during continuous drinking of a low-density barium suspension. It has been shown that rings with a maximal luminal diameter of more than 20 mm rarely cause symptoms, whereas rings with a maximal diameter of less than 13 mm usually are symptomatic (Schatzki 1963).

5.2.5. Diverticula

Esophageal diverticula may be classified as *pulsion* or *traction* diverticula. The more common pulsion diverticula result from esophageal dysmotility with increased intraluminal pressures in the esophagus, whereas traction diverticula are caused by scarring in the soft tissues surrounding the esophagus. Diverticula most commonly occur in the region of the pharyngoesophageal junction (i.e., Zenker's diverticulum), midesophagus, and distal esophagus above the gastroesophageal junction (i.e., epiphrenic diverticulum).

5.2.5.1. Pulsion Diverticula

Pulsion diverticula tend to be located in the distal esophagus and are often associated with fluoroscopic or manometric evidence of esophageal dysmotility. These diverticula usually are detected as incidental findings in patients who have no esophageal symptoms. However, a large epiphrenic diverticulum adjacent to the gastroesophageal junction may fill with debris, causing dysphagia, regurgitation, or aspiration. Pulsion diverticula appear on barium studies as rounded outpouchings from the esophageal lumen that have wide necks. They often do not empty completely when the esophagus collapses and may be associated with other radiologic findings of esophageal motor dysfunction (Levine 2000).

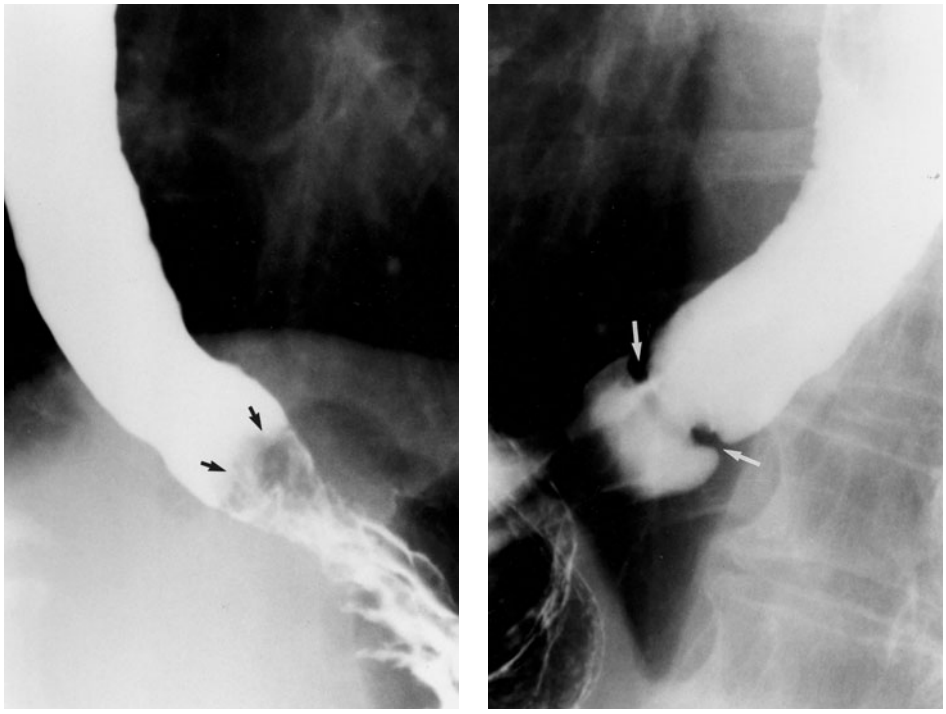

Fig. 5.2.23 Food impaction caused by Schatzki ring. **a** Initial view shows polypoid defect in distal esophagus with irregular meniscus superiorly (*arrows*) due to impacted meat in this region. **b** After impaction has been relieved, repeat view shows underlying Schatzki ring as cause of impaction. Note smooth, symmetric ring-like constriction (*arrows*) at gastroesophageal junction above small hiatal hernia

5.2.5.2. Traction Diverticula

Traction diverticula occur in the midesophagus and usually are caused by scarring from tuberculosis or histoplasmosis involving subcarinal or perihilar lymph nodes. Traction diverticula are true diverticula containing all layers of the esophageal wall and therefore maintain their elastic recoil. As a result, they tend to empty their contents when the esophagus collapses at fluoroscopy (Levine 2000). Traction diverticula often have a triangular or tented appearance caused by traction on the diverticulum by the fibrotic process in the adjacent mediastinum (Fig. 5.2.24). Thus, it often is possible to distinguish traction diverticula from pulsion diverticula on barium studies.

Fig. 5.2.24 Traction diverticulum. Single-contrast view shows triangular outpouching (*large arrow*) from midesophagus. Note surgical clip (*small arrow*) in adjacent mediastinum. The diverticulum resulted from postsurgical scarring in this region

5.2.6. Varices

Esophageal varices can be classified as *uphill* or *downhill*. Uphill varices are caused by portal hypertension with increased pressure in the portal venous system transmitted upwards via dilated esophageal collaterals to the superior vena cava. In contrast, downhill varices are caused by superior vena cava obstruction with downward flow via dilated esophageal collaterals to the portal venous system and inferior vena cava. Uphill varices are much more common than downhill varices.

5.2.6.1. Uphill Varices

Uphill esophageal varices develop as a result of portal hypertension or other causes of portal venous obstruction. Varices appear on barium studies as serpiginous or tortuous longitudinal filling defects in the distal half of the thoracic esophagus (Fig. 5.2.25). They are best seen on mucosal relief views of the collapsed esophagus using a high-density barium suspension to increase mucosal adherence (Cockerill et al. 1976). Even with proper technique, esophageal varices may be seen as a transient finding due to periodic filling and emptying of the varices with esophageal peristalsis or distention. The differential diagnosis for varices includes submucosally infiltrating esophageal carcinomas (varicoid carcinomas) and esophagitis with thickened folds due to submucosal inflammation and edema.

Esophageal varices are characterized on CT by a thickened, lobulated esophageal wall containing

Fig. 5.2.25 Esophageal varices. Double-contrast view shows serpiginous longitudinal defects etched in white in lower third of esophagus due to uphill esophageal varices

tubular structures that enhance markedly after intravenous administration of contrast material. Additional varices may be seen elsewhere in the abdomen at other sites of communication between the portal and systemic venous circulations. Angiography of the celiac or superior mesenteric arteries can be used to confirm the presence of extensive collateral flow and varix formation in and around the distal esophagus. However, the need for portal venography for presurgical planning of portosystemic shunts has decreased with the widespread use of transjugular intrahepatic portosystemic shunting (TIPS) procedures.

5.2.6.2. Downhill Varices

The most common cause of downhill varices is bronchogenic carcinoma with mediastinal metastases and superior vena cava obstruction. Additional causes include other primary or metastatic tumors involving the mediastinum, mediastinal irradiation, sclerosing mediastinitis, substernal goiter, and central catheter-related thrombosis of the superior vena cava. Most patients with downhill varices present clinically with superior vena cava syndrome and many have melena and iron-deficiency anemia from variceal bleeding.

Downhill varices typically appear as serpiginous longitudinal filling defects which, unlike uphill esophageal varices, are confined to the upper or mid-esophagus. Chest radiographs or CT often reveal the underlying cause of superior vena cava obstruction. Venography may also be performed to confirm the presence of superior vena cava obstruction.

5.2.7. Foreign Body Impactions

In adults, esophageal foreign body impactions most commonly are caused by inadequately chewed pieces of meat (Nandi and Ong 1978). Most of these foreign bodies pass spontaneously into the stomach, but 10-20% require some form of therapeutic intervention. Although the risk of perforation is less than 1% during the first 24 hours, this risk increases significantly after 24 hours because of ischemia and pressure necrosis at the site of impaction (Barber et al. 1984). Affected individuals typically present with acute onset of dysphagia, substernal chest pain, and/or a foreign body sensation.

Contrast studies often are performed in patients with suspected food impaction to confirm the presence of obstruction and determine its level and also to rule out esophageal perforation. An impacted food bolus typically appears as a polypoid defect with an irregular meniscus superiorly (Fig. 5.2.23a). Because of the degree of obstruction, it may be difficult to assess the underlying esophagus at the time of impaction. It therefore is prudent to perform a follow-up barium study after the impaction has been relieved to determine if this impaction was caused by a pathologic area of narrowing. The most common causes are Schatzki rings (Fig. 5.2.23b) and peptic strictures. Rarely, food impactions may even result from giant thoracic osteophytes impinging on the esophagus (Underberg-Davis and Levine 1991; Levine 2000).

Intravenous glucagon may be used to relax the lower esophageal sphincter and facilitate passage of an impacted bolus from the distal esophagus into the stomach (Trenkner et al. 1983). Effervescent agents may also help to distend the esophagus, promoting passage of the bolus (Robbins and Shortsleeve 1994). Over time, however, pressure necrosis may develop at the site of impaction, so gas-forming agents should be avoided if the obstruction has been present more than 24 hours to minimize the risk of perforation. Combination therapy with intravenous glucagon, an effervescent agent, and water has been shown to have a success rate of 70% in relieving esophageal food impactions without need for endoscopic intervention (Robbins and Shortsleeve 1994).

5.2.8. Fistulas

Esophageal-airway fistulas usually result from direct invasion of the tracheobronchial tree by advanced esophageal cancer (Fig. 5.2.26). Such fistulas have been reported in 5–10% of patients with esophageal cancer, often occurring after treatment with radiation therapy (Fitzgerald et al. 1981; Little et al. 1984). Other causes of esophageal-airway fistulas include esophageal instrumentation, trauma, foreign bodies, and surgery (Vasquez et al. 1988). Affected individuals typically present with violent episodes of coughing and choking during deglutition. When an esophageal-airway fistula is suspected on clinical grounds, barium should be used instead of hyperosmolar water-soluble contrast agents which may cause severe pulmonary edema if a fistula is present.

Esophagopleural fistulas may be caused by esophageal carcinoma, radiation therapy, surgery, or instrumentation. Such patients may present with a pleural effusion, pneumothorax, or hydropneumothorax. When an esophagopleural fistula is suspected, the

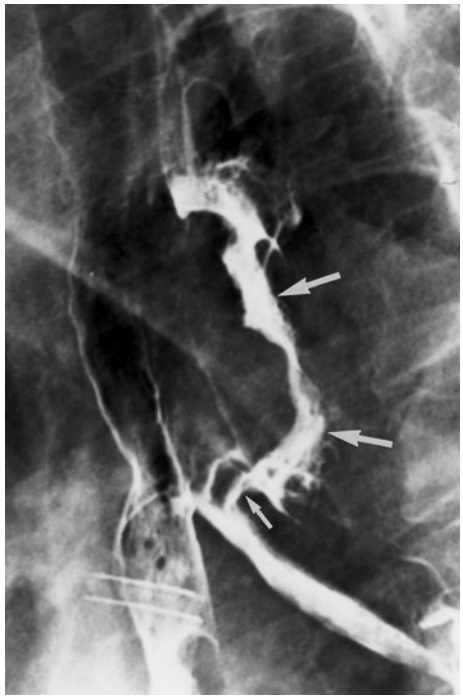

Fig. 5.2.26 Advanced esophageal carcinoma with esophagobronchial fistula. An infiltrating carcinoma (*large arrows*) is seen in midthoracic esophagus with barium in airway as result of esophagobronchial fistula (*small arrow*)

presence and location of the fistula can be confirmed by a study with water-soluble contrast media.

Aortoesophageal fistulas are extremely rare but are associated with a high mortality rate. Such fistulas may be caused by a ruptured aortic aneurysm, aortic dissection, infected aortic graft, swallowed foreign body, or esophageal cancer (Baron et al. 1981; Hollander and Quick 1991). Patients with aortoesophageal fistulas may present with an initial episode of arterial hematemesis followed by a variable latent period, before experiencing massive hematemesis, exsanguination, and death (Baron et al. 1981). Oral studies with water-soluble contrast agents are unlikely to show the fistula because of high aortic pressures, whereas contrast aortography may fail to show the fistula because of occlusion of the fistulous tract by thrombus (Baron et al. 1981).

5.2.9. Perforation

If untreated, perforation of the esophagus is associated with a mortality rate of nearly 100% because of a fulminant mediastinitis that occurs in these patients. Early diagnosis therefore is critical. Endoscopy is the most common cause of esophageal perforation, accounting for more than 75% of cases. Other causes include foreign bodies, food impactions, penetrating and blunt trauma, and spontaneous esophageal perforation due to a sudden, rapid increase in intraluminal esophageal pressure (Boerhaave's syndrome).

Cervical esophageal perforation may be manifested on plain films by subcutaneous emphysema, retropharyngeal air (Fig. 5.2.27a), and pneumomediastinum. Lateral films of the neck may also show widening of the prevertebral space or a retropharyngeal abscess containing loculated gas and/or air-fluid levels. In contrast, thoracic esophageal perforation may be associated with pneumomediastinum, mediastinal widening, and a pleural effusion or hydropneumothorax (Parkin 1973) (O'Connell 1967). In the proper setting, the presence of mediastinal gas on CT should be highly suggestive of esophageal perforation, whereas other findings such as pleural effusion or mediastinal fluid are less specific (White et al. 1993). However, CT is unreliable for determining the site of perforation.

Esophagography often is performed on patients with suspected esophageal perforation (Figs. 5.2.27b and 5.2.28). Although barium is the most sensitive contrast agent for detecting small leaks, it potentially can cause a granulomatous reaction in the mediastinum. In contrast, water-soluble agents do not incite a mediastinal reaction and are readily absorbed from the mediastinum if a leak is present. However, water-soluble contrast agents are less radiopaque than barium and can miss as many as 15–50% of esophageal perforations (Buecker et al. 1997). These agents can also cause severe pulmonary edema if aspirated into the lungs. It therefore is recommended that the examination be repeated with barium to detect subtle leaks if the initial study with water-soluble contrast media shows no evidence of perforation.

5.2.10. Congenital Esophageal Stenosis

Congenital esophageal stenosis occasionally may be diagnosed in adults, typically men with a life-long history of dysphagia and recurrent food impactions (McNally et al. 1993). The stenotic segment may appear on barium studies as a midesophageal stricture with distinctive ring-like indentations resembling tracheal rings (Fig. 5.2.29) (Mauro and Jaques 1981). In the appropriate setting, this appearance therefore should suggest a diagnosis of congenital esophageal stenosis.

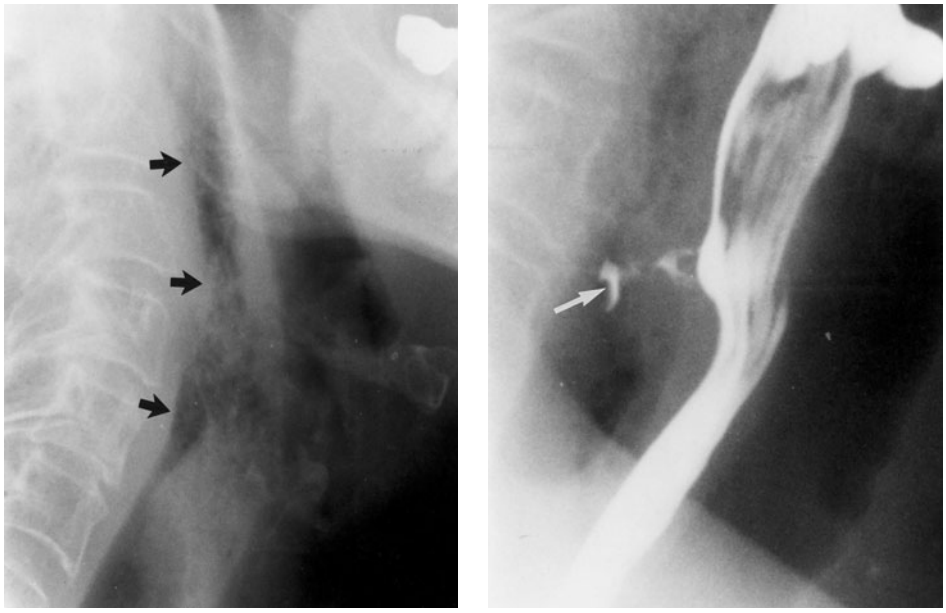

Fig. 5.2.27 Perforation of cervical esophagus. **a** Lateral view of neck shows mottled gas (*arrows*) in retropharyngeal space. **b** Subsequent study with water-soluble contrast material shows small, sealed-off perforation (*arrow*) from posterior aspect of pharyngoesophageal junction. This perforation was caused by traumatic endoscopy

Fig. 5.2.28 Perforation of thoracic esophagus. Study with water-soluble contrast material shows focal extravasation of contrast from right lateral wall of lower esophagus (*small arrows*) into collection (*large arrows*) in adjacent mediastinum. This perforation occurred as a complication of endoscopic dilatation

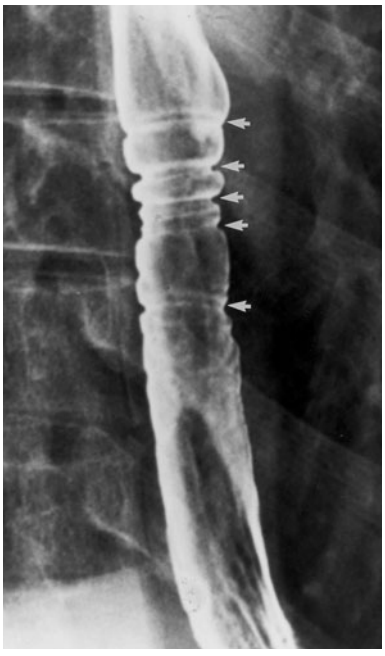

Fig. 5.2.29 Congenital esophageal stenosis. Double-contrast view shows mild stricture in midesophagus with distinctive ring-like indentations (*arrows*) in region of stricture. This appearance is characteristic of congenital esophageal stenosis

References

Agha FP and Keren DF (1985) Spindle-cell squamous carcinoma of the esophagus: a tumor with biphasic morphology. Am J Roentgenol 145:541–545

Appelqvist P and Salmo M (1980) Lye corrosion carcinoma of the esophagus: a review of 63 cases. Cancer 45:2655–2658

Banfield WJ and Hurwitz AL (1974) Esophageal stricture associated with nasogastric intubation. Arch Intern Med 134: 1083–1086

Barber GB, Peppercorn MA, Ehrlich C, and Thurer R (1984) Sophageal foreign body perforation: report of an unusual case and review of the literature. Am J Gastroenterol 79:509–511

Baron RL, Koehler RE, Gutierrez FR, Forrest JV, and Weyman PJ (1981) Clinical and radiographic manifestations of aortoesophageal fistulas. Radiology 141:599–605

Bleshman MH, Banner MP, Johnson RC, and DeFord JW (1978) The inflammatory esophagogastric polyp and fold. Radiology 128:589–593

Bova JG, Dutton NE, Goldstein HM, and Hoberman LJ (1987) Medication-induced esophagitis: diagnosis by double-contrast esophagography. Am J Roentgenol 148:731–732

Buecker A, Wein BB, Neuerburg JM, and Guenther RW (1997) Esophageal perforation: comparison of use of aqueous and barium-containing contrast media. Radiology 202:683–686

Carlyle DR, Goldstein HM, Wallace S, Medellin H, and Ventemiglia RA (1976) Azygography in the pretreatment evaluation of esophageal carcinoma. Br J Radiol 49:670–677

Carnovale RL, Goldstein HM, Zornoza J, and Dodd GD (1977) Radiologic manifestations of esophageal lymphoma. Am J Roentgenol 128:751–754

Carter R and Brewer LA (1975) Achalasia and esophageal carcinoma studies in early diagnosis for improved surgical management. Am J Surg 130:114–120

Chen YM, Ott DJ, Gelfand DW, and Munitz HA (1985) Multiphasic examination of the esophagogastric region for strictures, rings, and hiatal hernia: evaluation of the individual techniques. Gastrointest Radiol 10:311–316

Chen YM, Gelfand DW, Ott DJ, and Munitz HA (1987) Natural progression of the lower esophageal mucosal ring. Gastrointest Radiol 12:93–98

Cockerill EM, Miller RE, Chernish SM, McLaughlin GC, and Rodda BE (1976) Optimal visualization of esophageal varices. Am J Roentgenol 126:512–523

Collazzo LA, Levine MS, Rubesin SE, and Laufer I (1997) Acute radiation esophagitis: radiographic findings. Am J Roentgenol 169:1067–1070

Creteur V, Laufer I, Kressel, HY, Caroline DF, Goren RA, Evers KA, Glick SN, and Gatenby RA (1983a) Drug-induced esophagitis detected by double-contrast radiography. Radiology 147:365–368

Creteur V, Thoeni RF, Federle MP, Cello JP, Moss AA, Ominsky SH, Goldberg HI, and Axel L (1983b) The role of single and double-contrast radiography in the diagnosis of reflux esophagitis. Radiology 147:71–75

Dafoe CS and Ross CA (1969) Acute corrosive esophagitis. Thorax 24:291–294

DiPalma JA, Prechter GC, and Brady CE (1984) X-ray-negative dysphagia: is endoscopy necessary? J Clin Gastroenterol 6: 409–411

Ekberg O and Nylander G (1983) Webs and web-like formations in the pharynx and cervical esophagus. Diagn Imaging 52:10–18

Fitzgerald RH, Bartles DM, and Parker EF (1981) Tracheoesophageal fistulas secondary to carcinoma of the esophagus. J Thorac Cardiovasc Surg 82:194–197

Gefter WB, Laufer I, Edell S, and Gohel VK (1981) Candidiasis in the obstructed esophagus. Radiology 138:25–28

Gershwind ME, Chiat H, Addei KA, and Ferraro LR (1978) Granular cell tumors of the esophagus. Gastrointest Radiol 2:327–330

Gilchrist AM, Levine MS, Carr RF, Saul SH, Katzka DA, Herlinger H, and Laufer I (1988) Barrett's esophagus: diagnosis by double-contrast esophagography. Am J Roentgenol 150: 97–102

Glanz I and Grunebaum M (1977) The radiological approach to leiomyoma of the esophagus with a long-term follow-up. Clin Radiol 28:197–200

Glick SN, Teplick SK, Goldstein J, Stead JA, and Zitomer N (1982) Glycogenic acanthosis of the esophagus. Am J Roentgenol 139:683–688

Glick SN, Teplick SK, Levine MS, and Caroline DF (1986) Gastric cardia metastasis in esophageal carcinoma. Radiology 160:627–630

Gloyna RE, Zornoza J, and Goldstein HM (1977) Primary ulcerative carcinoma of the esophagus. Am J Roentgenol 129:599–600

Goldstein HM, Rogers LF, Fletcher GH, and Dodd GD (1975) Radiological manifestations of radiation-induced injury to the normal upper gastrointestinal tract. Radiology 117: 135–140

Goldstein HM, Zornoza J, and Hopens T (1981) Intrinsic diseases of the adult esophagus: benign and malignant tumors. Semin Roentgenol 16:183–197

Graham J, Barnes M, and Rubentein AS (1959) The nasogastric tube as a cause of esophagitis and stricture. Am J Surg 98: 116–199

Haulk AA and Sugar AM (1991) Candida esophagitis. Adv Intern Med 36:307–318

Hollander JE and Quick G (1991) Aortoesophageal fistula: a comprehensive review of the literature. Am J Med 91: 279–287

Hu C, Levine MS, and Laufer I (1997) Solitary ulcers in reflux esophagitis: radiographic findings. Abdom Imaging 22:5–7

Johnston J and Helwig EB (1981) Granular cell tumors of the gastrointestinal tract and perianal region: a study of 74 cases. Dig Dis Sci 26:807–816

Kressel HY, Glick SN, Laufer I, and Banner M (1981) Radiologic features of esophagitis. Gastrointest Radiol 6:103–108

Laufer I (1982) Radiology of esophagitis. Radiol Clin North Am 20:687–699

Levine MS (1994) Reflux esophagitis and Barrett's esophagus. Semin Roentgenol 29:332–340

Levine MS (2000) Miscellaneous abnormalities of the esophagus. In: Gore RM and Levine MS (eds) Textbook of gastrointestinal radiology. WB Saunders, Philadelphia, pp 465–483

Levine MS and Goldstein HW (1984) Fixed transverse folds in the esophagus: a sign of reflux esophagitis. Am J Roentgenol 143:275–278

Levine MS and Halvorsen RA (2000) Carcinoma of the esophagus. In: Gore RM and Levine MS (eds) Textbook of gastrointestinal radiology. WB Saunders, Philadelphia, pp 465–483

Levine MS, Laufer I, Kressel HY, and Friedman HM (1981) Herpes esophagitis. Am J Roentgenol 136:863–866
Levine MS, Kressel HY, Caroline DF, Laufer I, Herlinger H, and Thompson JJ (1983) Barrett esophagus: reticular pattern of the mucosa. Radiology 147:663–667
Levine MS, Caroline D, Thompson JJ, Kressel HY, Laufer I, and Herlinger H (1984) Adenocarcinoma of the esophagus: relationship to Barrett mucosa. Radiology 150:305–309
Levine MS, Macones AJ, and Laufer I (1985) Candida esophagitis: accuracy of radiographic diagnosis. Radiology 154:581–587
Levine MS, Dillon EC, Saul SH, and Laufer I (1986) Early esophageal cancer. Am J Roentgenol 146:507–512
Levine MS, Woldenberg R, Herlinger H, and Laufer I (1987) Opportunistic esophagitis in AIDS: radiographic diagnosis. Radiology 165:815–820
Levine MS, Loevner LA, Saul SH, Rubesin SE, Herlinger H, and Laufer I (1988) Herpes esophagitis: sensitivity of double-contrast esophagography. Am J Roentgenol 151:57–62
Levine MS, Fisher AR, Rubesin SE, Laufer I, Herlinger H, and Rosato EF (1991a) Complications after total gastrectomy and esophagojejunostomy: radiologic evaluation. Am J Roentgenol 157:1189–1194
Levine MS, Loercher G, Katzka DA, Herlinger H, Rubesin SE, and Laufer I (1991b) Giant, human immunodeficiency virus-related ulcers in the esophagus. Radiology 180:323–326
Levine MS, Herman JB, and Furth EE (1995) Barrett's esophagus and esophageal adenocarcinoma: the scope of the problem. Abdom Imaging 20:291–298
Levine MS, Buck JL, Pantongrag-Brown L, Buetow PC, Hallman JR, and Sobin LH (1996) Fibrovascular polyps of the esophagus: clinical, radiographic, and pathologic findings in 16 patients. Am J Roentgenol 166:781–787
Levine MS, Chu P, Furth EE, Rubesin SE, Laufer I, and Herlinger H (1997) Carcinoma of the esophagus and esophagogastric junction: sensitivity of radiographic diagnosis. Am J Roentgenol 168:1423–1426
Little AG, Ferguson MK, DeMeester TR, Hoffman PC, and Skinner DB (1984) Esophageal carcinoma with respiratory tract fistula. Cancer 53:1322–1328
Livingston EM and Skinner, DB (1985) Tumors of the esophagus. In: Berk JE (ed) Gastroenterology. WB Saunders, Philadelphia, pp 818–850
Macpherson RI (1993) Gastrointestinal tract duplications: clinical, pathologic, etiologic, and radiologic considerations. Radiographics 13:1063–1080
Mauro MA and Jaques PF (1981) Granular-cell tumors of the esophagus and common bile duct. J Can Assoc Radiol 32: 254–256
McNally PR, Lemon JC, Goff JS, and Freeman SR (1993) Congenital esophageal stenosis presenting as noncardiac, esophageal chest pain. Dig Dis Sci 38:369–373
Miller BJ, Murphy F, and Lukie BE (1978) Squamous cell papilloma of esophagus. Can J Surg 21:538–540
Nandi P and Ong GB (1978) Foreign body in the esophagus: review of 2394 cases. Br J Surg 65:5–9
O'Connell ND (1967) Spontaneous rupture of the esophagus. Am J Roentgenol Radium Ther Nucl Med 99:186–203
Parkin GJ (1973) The radiology of perforated esophagus. Clin Radiol 24:324–332
Pera M, Cameron AJ, Trastek VF, Carpenter HA, and Zinsmeister AR (1993) Increasing incidence of adenocarcinoma of the esophagus and esophagogastric junction. Gastroenterology 104:510–513
Pope CE (1994) Acid-reflux disorders. N Engl J Med 331: 656–660
Rafal RB and Markisz JA (1991) Magnetic resonance imaging of an esophageal duplication cyst. Am J Gastroenterol 86: 1809–1811
Robbins AH, Vincent ME, Saini M, and Schimmel EM (1978) Revised radiologic concepts of the Barrett esophagus. Gastrointest Radiol 3:377–381
Robbins MI and Shortsleeve MJ (1994) Treatment of acute esophageal food impaction with glucagon, an effervescent agent, and water. Am J Roentgenol 162:325–328
Rubesin S, Herlinger H, and Sigal H (1985) Granular cell tumors of the esophagus. Gastrointest Radiol 10:11–15
Ryan JM, Kelsey P, Ryan BM, and Mueller PR (1998) Alendronate-induced esophagitis: case report of a recently recognized form of severe esophagitis with esophageal stricture-radiographic features. Radiology 206:389–391
Sam JW, Levine MS, Rubesin SE, and Laufer I (2000) The "foamy" esophagus: a radiographic sign of Candida esophagitis. Am J Roentgenol 174:999–1002
Sandvik AK, Aase S, Kveberg KH, Dalen A, Folvik M, and Naess O (1996) Papillomatosis of the esophagus. J Clin Gastroenterol 22:35–37
Schatzki R (1963) The lower esophageal ring. Long term follow-up of symptomatic and asymptomatic rings. Am J Roentgenol 90:805
Schatzki R and Gary JE (1956) The lower esophageal ring. Am J Roentgenol 75:246
Seremetis MG, Lyons WS, deGuzman VC, and Peabody JW Jr (1976) Leiomyomata of the esophagus. An analysis of 838 cases Cancer 38:2166–2177
Shortsleeve MJ and Levine MS (1992) Herpes esophagitis in otherwise healthy patients: clinical and radiographic findings. Radiology 182:859–861
Sor S, Levine MS, Kowalski TE, Laufer I, Rubesin SE, and Herlinger H (1995) Giant ulcers of the esophagus in patients with human immunodeficiency virus: clinical, radiographic, and pathologic findings. Radiology 194:447–451
Spechler SJ, Robbins AH, Rubins HB, Vincent ME, Heeren T, Doos WG, Colton T, and Schimmel EM (1984) Adenocarcinoma and Barrett's esophagus An overrated risk? Gastroenterology 87:927–933
Takashima S, Takeuchi N, Shiozaki H, Kobayashi K, Morimoto S, Ikezoe J, Tomiyama N, Harada K, Shogen K, and Kozuka T (1991) Carcinoma of the esophagus: CT vs. MR imaging in determining respectability. Am J Roentgenol 156: 297–302
Taylor AJ, Stewart ET, and Dodds WJ (1990) The esophageal "jet" phenomenon revisited. Am J Roentgenol 155: 289–290
Totten R, Stout AP, and Humphreys GH (1953) Benign tumors and cysts of the esophagus. J Thorac Surg 25:606–622
Trenkner SW, Maglinte DD, Lehman GA, Chernish SM, Miller RE, and Johnson CW (1983) Esophageal food impaction: treatment with glucagon. Radiology 149:401–403
Underberg-Davis S and Levine MS (1991) Giant thoracic osteophyte causing esophageal food impaction. Am J Roentgenol 157:319–320
Vasquez RE, Landay M, Kilman WJ, Estrera A, and Schreiber T (1988) Benign esophagorespiratory fistulas in adults. Radiology 167:93–96
Vilgrain V, Mompoint D, Palazzo L, Menu Y, Gayet B, Ollier P, Nahum H, and Fekete F (1990) Staging of esophageal carci-

noma: comparison of results with endoscopic sonography and CT. Am J Roentgenol 155:277–281
White CS, Templeton PA, and Attar S (1993) Esophageal perforation: CT findings. Am J Roentgenol 160:767–770
Whitman GJ and Borkowski GP (1989) Giant fibrovascular polyp of the esophagus: CT and MR findings. Am J Roentgenol 152:518–520
Yamamoto AJ, Levine MS, Katzka DA, Furth EE, Rubesin SE, and Laufer I (2001) Short-segment Barrett's esophagus: findings on double-contrast esophagography in 20 patients. Am J Roentgenol 176:1173–1178
Zeabart LE, Fabian J, and Nord HJ (1979) Squamous papilloma of the esophagus: a report of 3 cases. Gastrointest Endosc 25:18–20

5.3 Esophageal Manometry and 24-Hour pH-Monitoring

Karin Aksglæde, Per Thommesen, Peter Funch-Jensen

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5.3.1 Esophageal Manometry

Manometry and pH monitoring are important tests in evaluation of patients suspected of having motility disorders and/or GORD, but we do not recommend these as first choice investigations. If a functional esophageal disease is suspected, a video-radiologic investigation based on physiological principles using bread-and-barium could often separate patients with normal manometry from patients with severe motility disorders i.e. DOS and achalasia (Nellemann et al. 2000). Furthermore, this radiologic method could demonstrate GOR in adults with a sensitivity of 52% and a specificity of 100%, thus reducing the number of patients referred to manometry and pH monitoring (Aksglæde et al. 1999).

Manometry is used to measure intraluminal pressure and pressure changes in the esophagus generated by contractions in the circular muscles. At present it is used in the clinical evaluation of esophageal contractile activity, although it has been discussed, whether the use of manometry should be an investigational technique or used as a clinical test.

K. Aksglæde, MD; P. Thommesen, MD, DMSc;
P. Funch-Jensen, MD
Motility Laboratory, Department of Radiology R, Aarhus University Hospital, Nørrebrogade 44, 8000 Aarhus C, Denmark

Esophageal manometry can be used in patients with suspected primary or secondary motility disorders. Secondary esophageal motility disorders are those occurring in patients having a generalised or systemic disease, i.e. systemic sclerosis or diabetes mellitus. Furthermore, manometry is often used pre-operatively before fundoplication (Kahrilas et al. 1994).

5.3.1.1 Technique

Esophageal pressures are measured directly with solid-state transducers, or indirectly with external transducers connected to a water-perfused system. The solid-state system measures pressures independently of body position and requires no external water supply and pumps, thus making it advantageous in long-term investigations. The latter is most often used for short-term manometric investigations of the esophagus.

The pressure catheters are introduced through a nostril and guided into the stomach. The probe is retracted through the lower esophageal sphincter (LOS) at a rate of 0.5–1.0 cm/s ("rapid pull-through technique") to measure the resting lower esophageal sphincter pressure (LOSP) in proportion to the fundic pressure. The relaxation is best measured using the "station pull-through technique", where the probe is pulled through the LOS at 0.5–1.0 cm at a time until recordings become stable, and relaxation is determined by wet or dry swallows. The catheter which is often supplied with three or more pressure channels spaced at 5 cm apart, is placed with the distal opening 5 cm above the LOS and a manometric study of the esophageal body is performed. Pressure amplitude, velocity, and duration of contractions are registered after dry, wet, and solid swallows. (Keren et al. 1992). Furthermore, the intra-esophageal baseline pressure can be determined during continuous swallowing. Measurement of the relaxation of the LOS is not always easy, but a gradual increase in base-line pressure during swill is seen in patients with incomplete opening of the LOS (Funch-Jensen et al. 2000).

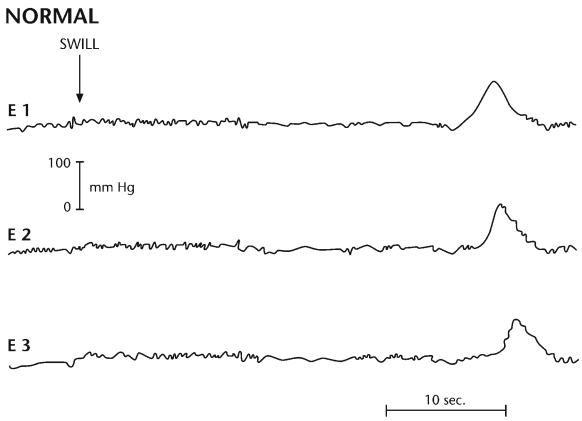


Fig. 5.3.1. Esophageal manometric recordings 15 cm (*E1*), 10 cm (*E2*), and 5 cm (*E3*) above the oral border of the lower esophageal sphincter (LOS) during continuous drinking (swill) in a patient with normal LOS relaxation, followed by a normal peristaltic contraction with normal amplitude and duration

5.3.1.2 Manometric Findings

The manometric tracings are classified according to generally accepted criteria:

1. Normal: Mean LOSP 10–30 mm Hg, and normal swallow-induced relaxation of LOS. In the body of the esophagus peristaltic pressure waves with a mean amplitude distally 30–110 mm Hg, and mean duration distally maximal 5.5 s. (Fig. 5.3.1)
2. Achalasia: Incomplete relaxation of LOS and aperistalsis and common cavity oscillations in the esophageal body (Fig. 5.3.2)
3. Diffuse esophageal spasms (DOS): Spontaneous or simultaneous repetitive broad-based contractions intermingled with normal peristaltic waves (Fig. 5.3.3)
4. Nutcracker esophagus: Peristaltic waves with amplitudes of more than 200 mm Hg
5. Hypomotility: Low amplitude contractions ≤30 mm Hg occurring peristaltically or non-peristaltically, with or without low resting LOSP
6. Non-specific esophageal motility disorder (NSOMD): Abnormal findings derived of classification according to the definitions above

5.3.1.3 Clinical Interpretation

Abnormal esophageal motility can potentially cause chest pain or dysphagia, or both. Interpretation of the clinical manometric result is rather simple and limited to a few possible pathologic observations, e.g.

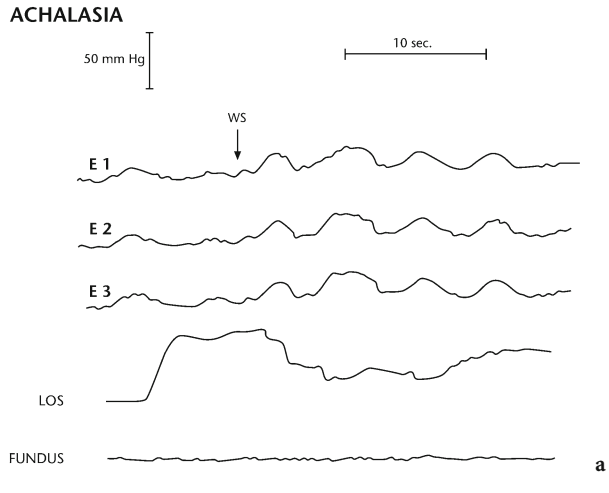


Fig. 5.3.2a,b. a Esophageal manometric recordings 15 cm (*E1*), 10 cm (*E2*), and 5 cm (*E3*) above the oral border of the lower esophageal sphincter (*LOS*) shows non-peristaltic common-cavity waves in the esophageal body during deglutition in patient with achalasia. Recording in LOS shows incomplete relaxation. **b** Esophageal manometric recordings during swill show a steady increase in base-line pressure in a patient with achalasia and incomplete LOS relaxation. This method can be used in patients where gastric intubations are impossible. Compare to Fig. 5.3.1

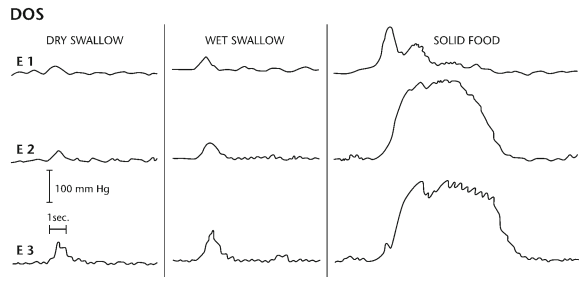


Fig. 5.3.3. The manometric signature after dry, wet, and solid swallow. Although pressure peaks are simultaneous during dry and wet swallows, the diagnosis of diffuse esophageal spasms (*DOS*) is only possible after the solid bolus

weak or absent peristalsis, disordered peristalsis, or impaired LOS relaxation.

Although manometry is sensitive in detecting esophageal motor disorders, it is often non-specific, except when achalasia or DOS is a diagnostic possibility (Kahrilas et al. 1994; Nelleman et al. 2000).

5.3.2 24-Hour pH Monitoring

Esophageal pH monitoring can document normal or abnormal esophageal acid exposure. Indications for prolonged esophageal pH monitoring include:

1. Patients with typical symptoms of gastro-esophageal reflux disease (GORD) and normal endoscopy
2. Patients with atypical symptoms of GORD (i.e. non-cardiac chest pain, pulmonary symptoms, hoarseness)
3. Prone to and subsequent to antireflux therapy (medical or surgical)

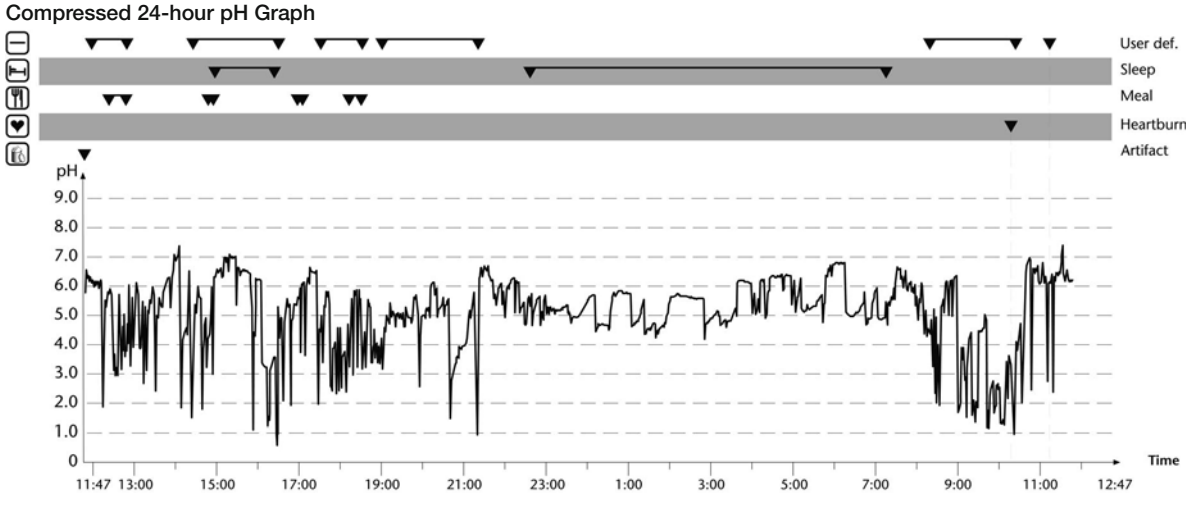
5.3.2.1 Technique

The basic equipment requirements for ambulatory esophageal pH studies include a portable data logger for data storage, a pH electrode, a computer and software for analysis of the pH data.

Modern data logger's used in esophageal ambulatory pH studies are lightweight, battery-powered units that can be worn by the patient on a waist belt or shoulder straps. The data logger also has an event marker that can be activated by the patient during the study to indicate the timing of symptoms, meals, and recumbency (sleep).The patient can also record these events on a diary card.

The probes are usually 2–4 mm in diameter, composed of antimony or glass, and an internal or external reference electrode. Also, each probe consists of single or multiple channels which can be customized for the individual laboratories.

The pH probe is passed through a nostril into the stomach to verify an acidic pH, and then positioned



Period Table

Item		Total	Upright	Supine	Meal	PostP	NONE	HrtBrn
Duration of period	(HH:MM)	24:00	13:52	10:08	01:21	09:07	08:28	00:04
Number of acid refluxes	(#)	139	136	4	22	85	96	1
Number of long acid refluxes	(#)	6	5	1	0	5	6	0
Longest acid reflux	(min)	31	31	20	1	31	31	4
Total time pH below 4.00	(min)	165	144	21	8	130	139	4
Fraction time pH below 4.00	**(%)**	**11.5**	**17.3**	**3.5**	**9.3**	**23.7**	**27.4**	**100.0**
Symptom Index	(%)	n/a	n/a	n/a	n/a	n/a	83.3	100.0

DeMeester score

Total score= 39.5 DeMeester normals: <14.72 (95th percentile)

Fig. 5.3.4. Compressed 24-h pH graph and period table. Fraction time of pH below 4 is accentuated

5 cm above the superior margin of the gastro-esophageal junction (GOJ), identified by manometry or radiography (Aksglæde et al. 1999; 2003). This probe positioning avoids possible probe displacement into the stomach due to swallow-induced esophageal shortening, which is not fully compensated by concomitant electrode oscillation during eating and talking (Aksglæde et al. 2003).

The length of the study should be at least16 h, thus allowing assessment of at least two post-prandial periods and overnight supine position.

5.3.2.2 Diet and Drugs While Performing the Study

Discontinuing prokinetic agents, drugs that neutralize acids, and drugs that have an influence on the LOSP 24–48 h prior to an examination is recommended. Proton pump inhibitors must be discontinued at least 72 h prior to examination due to the long half-life. During the examination the patient can eat a normal diet excluding acid food and beverage and physical activity need not be restricted.

5.3.2.3 Evaluation of pH Monitoring

Acid gastro-esophageal reflux (GOR) episodes are defined as periods with pH ≤ 4. To obtain a global assessment, several parameters are often used: The percentage of time pH ≤ 4 for the whole investigation time (reflux index), percentage of time pH ≤ 4 in the supine and upright periods, the number of reflux episodes, number of episodes lasting more than 5 min, and the longest reflux episode.

Johnson and DeMeester (1986) devised a scoring system based on these six parameters to calculate the degree in which reflux patterns differ between individuals (Fig. 5.3.4). However, in order to discriminate between physiological and pathological gastro-esophageal reflux in adults, the most useful parameter is the Reflux Index, with normal values varying from 3.9% to 7.2% (Kahrilas and Quigley 1996). We have adopted Reflux Index < 5% as the upper normal limit.

References

Aksglæde K, Funch-Jensen P, Thommesen P (1999) Radiological demonstration of gastroesophageal reflux. Diagnostic value of barium and bread studies compared with 24-h pH monitoring. Acta Radiol 40:652–655

Aksglæde K, Funch-Jensen P, Thommesen P (2003) Which is the better method for location of the gastro-esophageal junction: radiography or manometry? Acta Radiol 44: 121–126

Aksglæde K, Funch-Jensen P, Thommesen P (2003) Intraoesophageal pH probe movement during eating and talking. A video-radiographic study. Acta Radiol 44:131–135

Funch-Jensen P, Aksglæde K, Thommesen P (2000) A new method for the detection of incomplete lower esophageal sphincter relaxation in patients with achalasia. Scand J Gastroenterol 35:349–352

Johnson LF, DeMeester TR (1986) Development of the 24-h intraesophageal pH monitoring composite scoring system. J Clin Gastroenterol 8(1): 52–58

Kahrilas PJ, Quigley EMM (1996) Clinical esophageal pH recording: a technical review for practice guideline development. Gastroenterology 110:1982–1996

Kahrilas PJ, Clouse RE, Hogan WJ (1994) An American Gastroenterological Association Medical Position Statement on the Clinical Use of Esophageal Manometry. Gastroenterology 107:1865–1884

Keren S, Argaman E, Golan M (1992) Solid swallowing versus water swallowing: manometric study of dysphagia. Dig Dis Sci 37(4):603–608

Nellemann H, Aksglæde K, Funch-Jensen P, Thommesen P, (2000) Bread and barium. Diagnostic value in patients with suspected primary esophageal motility disorders. Acta Radiol 41:145–150

5.4 Intervention on the Esophagus

David J. Ott, Benoit C. Pineau, Michael Y. Chen

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5.4.1 Introduction

In the past, the evaluation of esophageal disease consisted mainly of diagnosis and possibly the use of surgical or radiation treatment. Major advancements in esophageal intervention have occurred in recent decades, especially with innovations with endoscopic techniques. The endoscopist will typically perform these interventional procedures, but imaging may be required during or after the procedure. Thus, the radiologist needs a thorough understanding of these newer techniques and knowledge of their radiographic appearances and potential complications. Interventional techniques in the esophagus center largely on the management of specific disorders or their complications. Important clinical conditions that may require evaluation and endoscopic management include treatment of esophageal hemorrhage, endoscopic mucosal ablation, removal of esophageal foreign bodies, pneumatic dilatation for treating achalasia, peptic stricture dilatation, and therapeutic or palliative management of esophageal malignancy, which often involves placement of metallic stents.

Major advancements in esophageal intervention, especially with the use of endoscopic techniques, have occurred in recent years (Edmundowicz 1999). The endoscopist will typically perform these interventional procedures, but imaging may be required during or after the procedure. Thus, the radiologist requires an understanding of these newer techniques and knowledge of their radiographic appearances and potential complications.

In this chapter, the evaluation and management of the following clinical conditions and procedures are discussed: 1) esophageal hemorrhage; 2) endoscopic mucosal ablation; 3) esophageal foreign bodies; 4) achalasia, especially the use of pneumatic dilatation; 5) peptic strictures; and 6) esophageal malignancy. The endoscopic procedures are reviewed and the role of imaging in assisting in the management of these disorders is discussed and illustrated. The newer metallic endoprostheses are emphasized because imaging is pivotal in their deployment, assessment, and subsequent evaluation.

5.4.2 Esophageal Hemorrhage

Endoscopy has become the primary method used for the diagnosis and potential treatment of upper gastrointestinal bleeding (Bass and Somberg 1998). Esophageal sources of hemorrhage are numerous but usually occur secondary to esophagitis, Mallory-Weiss tears, varices, or malignancies. Mallory-Weiss tears and varices are the most common causes of esophageal bleeding. The Mallory-Weiss tear is a longitudinal mucosal injury located at the esophagogastric junction that may follow an episode of vomiting with hematemesis. Esophageal varices are

D. J. Ott, MD., FACR, FACG
Department of Radiology, Wake Forest University School of Medicine, Medical Center Boulevard, Winston-Salem, NC 2715, USA
B. C. Pineau, MD, MSc (Epid.), FRCPC
Department of Medicine, Department of Radiology, Wake Forest University School of Medicine, Medical Center Boulevard, Winston-Salem, NC 2715, USA
M. Y. Chen, MD
Department of Radiology, Wake Forest University School of Medicine, Medical Center Boulevard, Winston-Salem, NC 2715, USA

most often caused by portal hypertension related to cirrhosis in western societies (Fig. 5.4.1). Thus, treatment of bleeding esophageal varices may be directed at the varices, alleviation of portal hypertension, or both.

Endoscopic techniques for control of esophageal bleeding include injection, thermal, and mechanical therapies (Edmundowicz 1999). Injection of submucosal or intramural epinephrine or sclerosants can be effective in controlling hemorrhage from a number of esophageal lesions, or may be combined with other therapies to improve hemostasis. Thermal therapies and coaptive technology with the heater probe or BICAP device are other options. The heater probe and BICAP device allow for effective and safe treatment of bleeding sites in the esophagus. This technology converts electrical energy to heat and causes a coaptive coagulation of the bleeding site when the device is applied with pressure. Hemoclips and variceal ligators are examples of mechanical therapies that may be used for nonvariceal or variceal hemorrhage.

Endoscopy plays a pivotal role in the diagnosis and management of esophageal variceal bleeding (Bass and Somberg 1998). Sclerotherapy involves injection of a sclerosant into or near the varix. Mechanical therapies can also be used to treat bleeding varices. Variceal ligation has emerged as the preferred method in the management of variceal hemorrhage; with the development of multiple banders, the need for an overtube or re-endoscopy has been eliminated. Balloon tamponade has been used for many years as an alternate method to temporarily control variceal bleeding by compression of the varices. Various balloon devices have been used but the four-lumen Minnesota tube is regarded as the safest because secretions above the esophageal balloon can be aspirated continuously.

Complications from these therapeutic modalities are variable and include ulceration with recurrent hemorrhage, stricture formation, perforation, mediastinitis, and pleural effusions (Agha 1984; Kochhar et al. 1992; Sharara and Rockey 2001; Subramanyam and Patterson 1986; Tihansky et al. 1984). Radiologic imaging has a limited role in the diagnosis of many of the esophageal causes of upper gastrointestinal bleeding although esophagitis, varices, and malignancies are often detected. Imaging may be more important in the evaluation of potential complications following these procedures (Fig. 5.4.2). In particular, a contrast esophagram can assess for perforation and stricture formation, although the latter complication has diminished with the newer therapies.

Fig. 5.4.1 Multiple linear varices in the lower esophagus

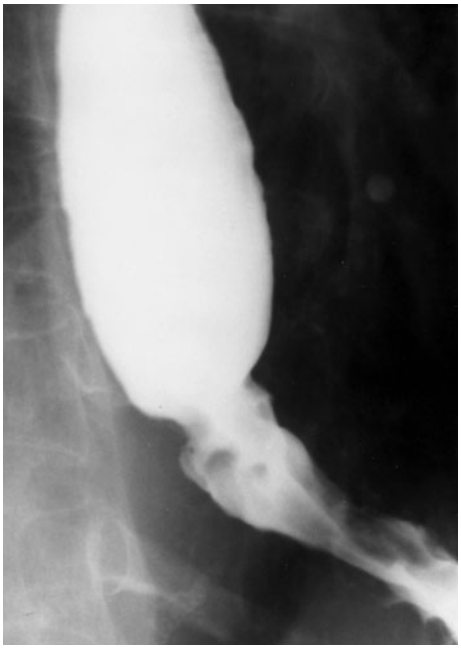


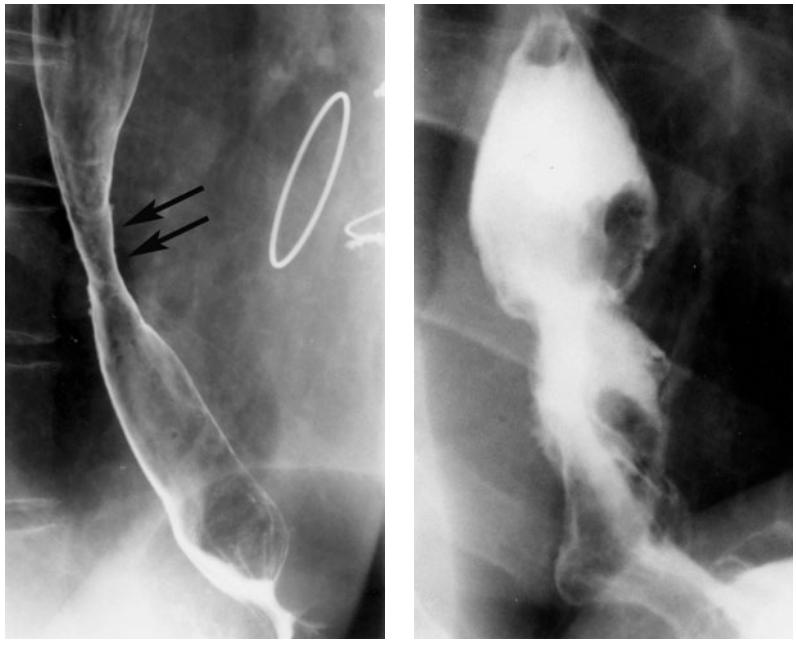
Fig. 5.4.2 Irregular, nodular narrowing at the lower end of the esophagus following endoscopic sclerotherapy. Repeat endoscopic examination showed a stricture and varices

5.4.3 Mucosal Ablation

Endoscopic resection of mucosal lesions of the esophagus is an emerging technique directed at the destruction of superficial lesions involving the mucosal and submucosal layers of the organ; endoscopic ultrasound may be used to confirm that a lesion is limited to these layers of the esophageal wall (Edmundowicz 1999). Mucosal ablation may be performed using chemical, thermal, or mechanical methods. Submucosal saline infiltration is often used to elevate a lesion from the muscularis propria, which is then removed by electrocautery snare. Other modalities employed to ablate superficial lesions include BICAP, heater probe, argon plasma coagulation, laser, and photodynamic therapy. The depth of injury must be limited to the inner layers of the esophageal wall to avoid perforation, which is the major complication.

Barrett's esophagus is a complication of gastroesophageal reflux disease and the columnar-lined mucosa in this disorder may develop dysplasia and progress to adenocarcinoma (Armstrong 1994; Cameron et al. 1995; Sampliner 1998). Thus, surveillance of patients with Barrett's esophagus has received increasing attention in recent years (Sampliner 1998; Soni et al. 2000). In patients with high-grade dysplasia, invasive malignancy may already be present or may develop in subsequent years. Therapeutic options include close endoscopic surveillance with mandatory mucosal biopsies (mucosal staining agents during endoscopy may also be used), a major surgical procedure involving esophagectomy with its associated morbidity and mortality, and the use of mucosal ablation techniques, which offer attractive alternative choices still under extensive investigation (Buttar et al. 2001; Ell et al. 2000; Sharma et al. 2001; Schnell et al. 2001; van den Boogert et al. 1999). The goal of these endoscopic therapies is to remove the columnar epithelium, and allow regeneration of squamous mucosa in an alkaline milieu (proton pump inhibitor medications, surgical fundoplication, or both). The use of these endoscopic ablative therapies to destroy the Barrett's epithelium remains, at present, largely experimental and will require further controlled studies before widespread applications can be recommended.

Complications related to mucosal ablation techniques will depend on the methods used, the types of lesions treated, and the experiences of the operators, and are similar to those discussed in the section on esophageal hemorrhage (Edmundowicz



a **b**

Fig. 5.4.3a,b. a Tapered stricture (*arrows*) in the mid-esophagus with lower normal-appearing segment consistent with Barrett's esophagus, which was confirmed at endoscopic examination. **b** Irregular nodularity and stricture in lower esophagus in a patient with Barrett's esophagus. Endoscopy with multiple biopsies showed complicating adenocarcinoma

1999). Potential complications include hemorrhage, ulceration, sinus tracts, perforation, fistula, stricture, and mediastinal processes (e.g. abscess). Radiologic imaging may be useful in pre-endoscopic evaluation of these patients (Fig. 5.4.3), assessment of immediate complications (e.g. esophageal perforation or mediastinal abscess), or post-procedural evaluation of patients with potential symptomatic complications (e.g. dysphagia and stricture formation) (Glick 1994; Levine et al. 1995).

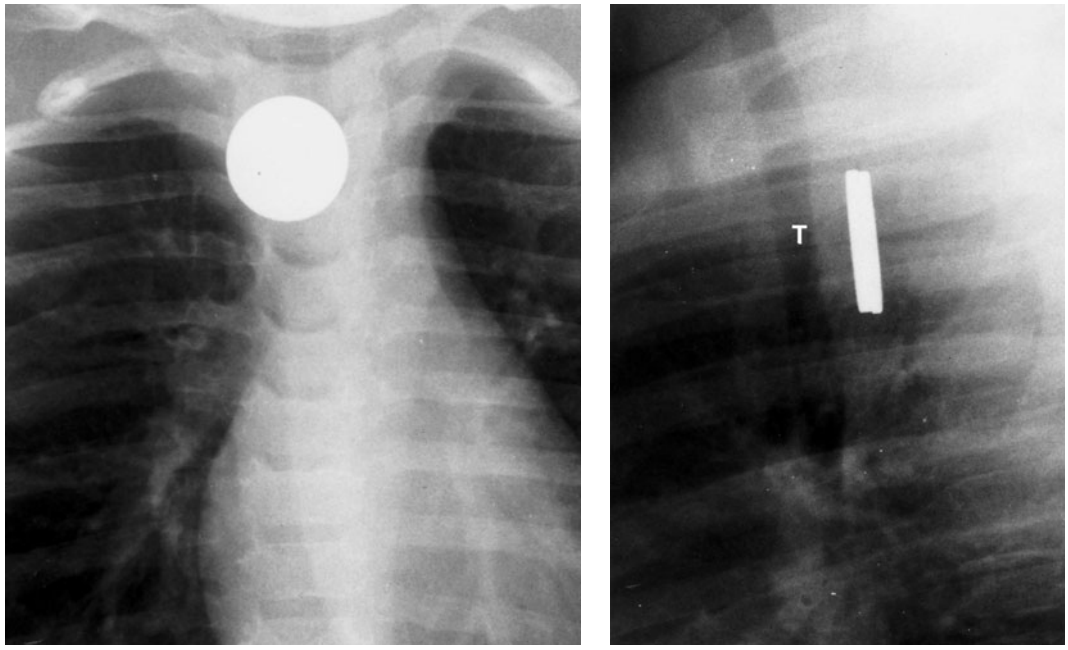
5.4.4. Esophageal Foreign Bodies

Foreign body ingestion remains a common problem and although the actual incidence is difficult to estimate, reports indicate that 1,500 or more deaths occur annually in the United States as a result of this problem (Yeaton and Peura 1999). Up to 90% of swallowed objects that reach the stomach will pass uneventfully through the gastrointestinal tract but the remaining minority may become lodged in the esophagus with subsequent risk to the patient. Potential complications from impacted esophageal foreign bodies include mucosal damage with bleeding, respiratory difficulty, luminal obstruction, perforation, and sinus tract and fistula formation to adjacent structures, which may lead to abscess, sepsis, and death. The goals of managing patients with potential esophageal foreign bodies include proper diagnosis, relief of symptoms, and prevention of complications.

Foreign bodies in the esophagus typically impact at physiologic or pathologic areas of narrowing with the most common site being the cervical inlet followed by the middle esophagus, and least likely the lower esophagus. In children, the foreign bodies seen most often include accidental ingestion of objects such as toys, coins, pins, batteries, and bones (Macpherson et al. 1996). Food impaction is more likely in adults although other objects, such as dentures or partial dental plates, can be swallowed (Longstreth et al. 2001). The type and nature of the object ingested, the duration of impaction, the location of the ingested material, and other factors will determine the approach in treating the patient. The diagnosis and treatment of esophageal foreign bodies is usually performed with radiologic or endoscopic techniques depending on these various considerations (Maglinte et al. 1995; Weinstock et al. 1999); surgical exploration is rarely needed unless perforation or subsequent extra-luminal complications occur.

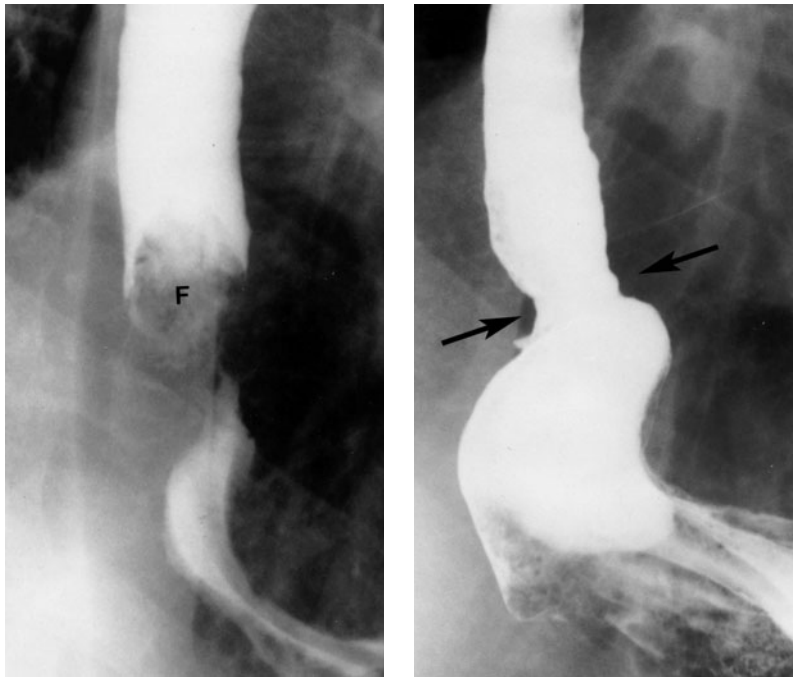
Radiologic imaging is usually used for the initial evaluation of the patient with suspected esophageal foreign body impaction. If an opaque object is suspected, such as a coin swallowed by a child, plain films are useful for detecting the foreign body and may also indicate the site of the impaction depending on the orientation of the coin (i.e. esophagus vs. trachea) (Fig. 5.4.4). Non-opaque foreign bodies in the esophagus can be evaluated with contrast examinations using water-soluble agents or barium suspensions; typically only several swallows of the contrast material are needed to indicate the presence, size, and location of a non-opaque foreign body. The choice between the use of a water-soluble or a barium agent may depend on the suspected location of the foreign body (e.g. barium is safer if aspiration risk is high in a cervical impaction), on the preference of the clinician or radiologist, or on the possible presence of complications, such as perforation. If endoscopy may be needed to remove the foreign body after its diagnosis by radiologic examination, a water-soluble contrast is preferable since an opaque barium suspension prevents adequate visualization of the impacted material making performance of the endoscopic procedure more difficult.

Depending on their structural nature (e.g. round and potentially mobile foreign bodies), radiologic removal of foreign bodies may be attempted (Fig. 5.4.5). For an acute distal food impaction, such as a piece of meat impacted in the lower esophagus, a radiologic approach using gas-crystals with water or a contrast material can be attempted (Kaszar-Seibert et al. 1990; Rice et al. 1983). The diagnosis of lower esophageal food impaction is first confirmed by radiological examination; the patient is then placed in the upright position and residual contrast material usually remains to outline the impacted material. The patient then ingests gas-producing crystals followed immediately by water or dilute contrast material; the generation of gas above the food often propels it into the stomach; the procedure can be repeated several times, as needed. The administration of glucagon intravenously (0.5–1.0 mg) may facilitate the passage of impacted food into the stomach using this technique (Colon et al. 1999; Trenkner et al. 1983). This method is successful and can be performed safely in most patients with lower esophageal food impactions. Fluoroscopic Foley catheter removal of foreign bodies, such as coins, in children is controversial, but is another radiologic therapeutic option available but used rarely (Macpherson et al. 1996). Typically, a deflated Foley catheter is placed distal to the foreign body, the balloon is inflated, and the catheter



a b

Fig. 5.4.4a,b. a Frontal radiograph of chest shows 'en-face' coin swallowed by a child with the appearance of the coin suggesting an esophageal location. **b** Lateral radiograph on the chest in the same patient shows the opposite orientation of the coin which is posterior to the trachea (*T*) and thus located in the esophagus



a b

Fig. 5.4.5a,b. a Food (*F*) impaction in the lower esophagus with small amount of contrast material progressing below the impaction. **b** Post-endoscopic removal of the food impaction, which was a meat bolus. Repeat contrast examination shows a stricture (*arrows*) and hiatal hernia

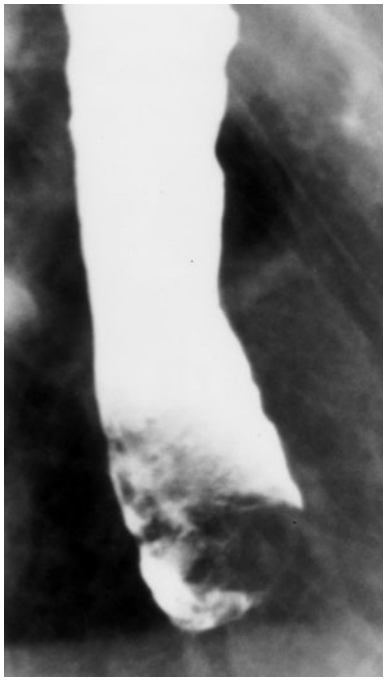
retracted orad dragging the coin (or other 'smooth' object) into the oral cavity.

Endoscopy can also be used for the diagnosis and more importantly the treatment of esophageal foreign bodies of various sizes and shapes, including sharp objects such as pins (Yeaton and Peura 1999). The endoscopic techniques employed to treat patients with foreign bodies in the esophagus will depend on several considerations including the type of material impacted, but the therapeutic modality of choice is often endoscopic extraction (Mamel et al. 1995; Saeed et al. 1990). An additional non-endoscopic technique, digestion of a meat bolus enzymatically with topical proteolytic agents, is no longer favored. Endoscopic accessories available for foreign body extraction include retrieval baskets, polyp grabbers, biopsy forceps, foreign body forceps, polypectomy snares, and overtubes (Fig. 5.4.6). Flexible (vs. rigid) endoscopic removal of a meat bolus is the preferred method of choice and can be performed much like standard endoscopic examinations. Coins, batteries, dental objects, and sharp and pointed objects require different and often more specific approaches (Fig. 5.4.7). The major risks of endoscopic removal include esophageal perforation, aspiration, and airway occlusion. Finally, following removal of a foreign body in the esophagus, regardless of the method used, a thorough radiographic or endoscopic examination should be performed to determine a potential underlying cause of the obstruction, such as a lower esophageal mucosal ring.

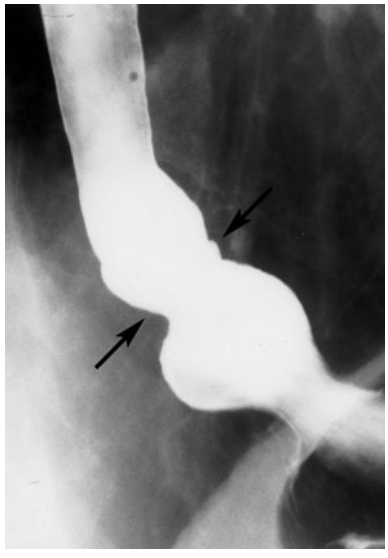
5.4.5 Achalasia

Achalasia is characterized by aperistalsis of the esophagus and lower esophageal sphincter dysfunction (Vaezi and Richter 1999). Treatment of achalasia involves reducing the functional obstruction of the sphincter to improve esophageal emptying and to relieve symptoms, such as dysphagia and regurgitation. Pneumatic dilatation of the esophagus (Fig. 5.4.8) and the modified Heller myotomy, which is now performed laparoscopically, are the most effective forms of therapy to treat achalasia (Imperiale et al. 2000; Vaezi and Richter 1999). Local injection of botulinum toxin is performed under endoscopic guidance and may offer temporary relief by reducing the lower esophageal sphincter tone (Kolbasnik et al. 1999; Prakash et al. 1999).

The 'open' Heller myotomy was the major alternative to pneumatic dilatation for the treatment of achalasia for many years but can now be done using laparoscopic techniques (Ackroyd et al. 2001; Cade 2000; Katilius and Velanovich 2001; Kumar et al. 1998). Similar to the open surgical methods, incision of the muscular layers of the esophagogastric junction over a short segment is performed laparoscopically. The addition of an anti-reflux wrap (partial or total) following the cardiomyotomy has been controversial, but a small percentage of patients will develop gastroesophageal reflux, which may be alleviated by use of an



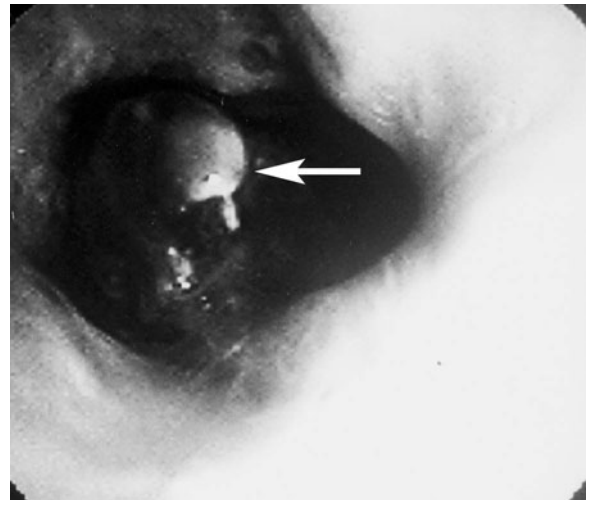
a



b

Fig. 5.4.6a,b. a Food impacted in the lower end of the esophagus. Radiographic attempts at dis-impaction of the food using gas crystals were unsuccessful. **b** Food impaction was removed using several endoscopic accessories. Repeat contrast examination shows a mild stricture (*arrows*) and hiatal hernia

a

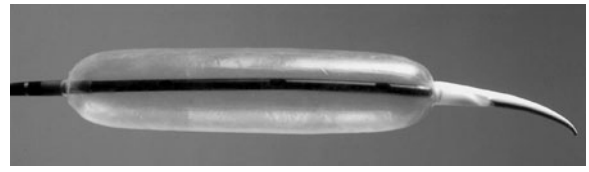


b

c

Fig. 5.4.7a,b. a. Patient swallowed a portion of a dental plate. Contrast esophagram shows a rectangular filling defect in the lower esophagus. **b**. Endoscopic image shows a tooth (*arrow*) attached to the dental plate. **c** Photograph of the dental plate after endoscopic retrieval; no complications occurred during the procedure

a



b

Fig. 5.4.8a,b. a Microvasive pneumatic dilator (Boston Scientific, Watertown, MA), which is a popular instrument used to treat achalasia. **b** Inflated balloon end of the same dilator; balloons are available in various sizes, ranging from 3.0 cm to 4.0 cm

anti-reflux procedure. Complications of this procedure may be immediate or delayed and include perforation at the operated site, gastro-esophageal reflux, and stricture; many of these potential complications can be evaluated with radiographic contrast examinations.

Pneumatic dilatation of the lower esophagus for the treatment of achalasia involves placement of an inflatable balloon across the esophagogastric junction using fluoroscopic guidance (Eckardt et al. 1997; Katz et al. 1998; Metman et al. 1999; Molina

et al. 1995; Ott et al. 1987; Ott et al. 1991; Vaezi et al. 1999). Larger pneumatic dilators, which measure 3.0–4.0 cm in diameter, are available for this procedure although a wide variety of different types of dilators were used in the past. The Rigiflex pneumatic dilator (Microvasive, Milford, MA) in the 30 or 35 mm sizes is commonly employed; a guidewire is placed into the stomach through an endoscope and its location verified fluoroscopically. The dilator is advanced over the guidewire and the balloon positioned at the esophagogastric junction. One or more inflations of the balloon can be performed; the impression from the lower esophageal sphincter is evident transiently by a constriction at the waist of the balloon but disappears quickly. Potential complications include malplacement of the dilator, mucosal injury with bleeding, intramural hemorrhage, and perforation.

Radiographic evaluation of the esophagus following pneumatic dilation is useful to detect serious complications, such as perforation (Fig. 5.4.9) which occurs in about 5% or less of patients using modern dilators (Eckardt et al. 1997; Metman et al. 1999; Molina et al. 1995; Ott et al. 1987; Ott et al. 1991; Vaezi et al. 1999). Although perforation of the esophagus is easily determined with contrast studies, especially if esophageal intubation is used, reports have conflicted on the value of the radiologic examination in predicting the clinical response of the patient. Parameters that have been studied include changes in the radiographic appearance of the esophagogastric region, rate of esophageal emptying using various contrast materials or radionuclide agents, type and size of the dilators and techniques used, inflation pressures generated and their duration, presence or absence of blood on the dilator, severity of chest pain during the procedure, and changes in manometric measurements, such as the lower esophageal sphincter pressure. Most investigations have shown that these parameters fail to predict the long term results after pneumatic dilatation.

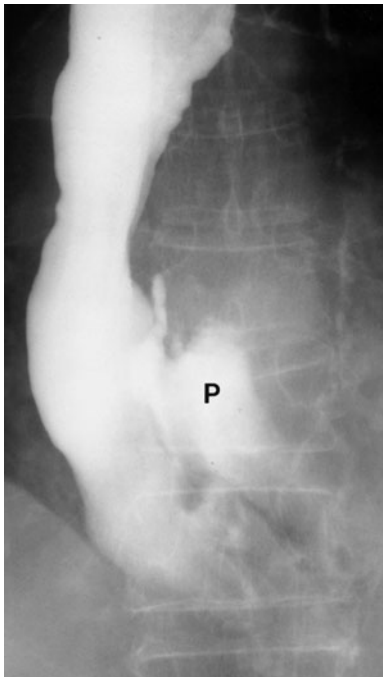


Fig. 5.4.9. Following pneumatic dilatation of the esophagus for achalasia, an intubated water-soluble contrast examination was performed which showed a large perforation (P) arising from the left lateral wall of the lower esophagus

Regarding radiographic changes following pneumatic dilatation, an increase in the caliber of the esophagogastric junction occurs commonly following this procedure. In a compilation of six reports using various types of dilators and techniques, the mean caliber of the esophagogastric junction increased from pre-dilation widths of 2.3–4.7 mm to post-dilation calibers of 4.8–10.0 mm (Ott and Chen 1998). Clinical improvement in these investigations was seen in 59% to 88% of the patients treated but no significant correlation was found between the symptomatic response of the patient and the change in caliber of the esophagogastric region. In addition, the contour of the post-dilated esophagus (i.e. smooth vs. irregular) and the status of esophageal emptying (i.e. immediate vs. delayed) do not correlate well in predicting the long term relief of patient symptoms (Ott et al. 1987; Ott et al. 1991). Thus, the main role of the radiographic examination used immediately following pneumatic dilatation for achalasia is to assess for serious complications, such as perforation, which may necessitate emergent surgery. Smaller intramural or confined perforations of the esophagus may also be seen in patients undergoing pneumatic dilatation and are often amenable to more conservative management.

5.4.6 Peptic Stricture

Focal narrowing of the esophagus results from a variety of inflammatory and neoplastic processes but peptic stricture of the lower esophagus is one of the most common causes (Edmundowicz 1999; Karasick and Lev-Toaff 1995; O'Connor and Richter 1999). Gastro-esophageal reflux disease can cause morphologic changes of reflux esophagitis that may progress to fibrotic narrowing and stricture for-

mation (Karasick and Lev-Toaff 1995; O'Connor and Richter 1999; Ott 1994). The typical peptic stricture is located at the esophagogastric junction and is usually found in the presence of hiatus hernia (Fig. 5.4.10). Differential considerations for focal narrowings in the lower esophagus include mucosal ring, malignant strictures, and intrinsic strictures due to other factors, such as sclerotherapy, radiation, or caustic ingestion; in addition, extrinsic processes or those arising from the stomach may involve or invade the esophagus and mimic peptic stricture. Thus, in patients with dysphagia and focal esophageal narrowing, radiographic and endoscopic examination is warranted in assessing these differential causes before therapy is initiated.

Once the diagnosis of peptic stricture is established and malignancy (and Barrett esophagus) has been excluded, therapeutic options can be considered. The clinical course of peptic stricture is usually chronic and progressive and the importance of controlling the underlying causes of this disease (i.e. anti-reflux therapy) must be understood. More aggressive treatment of peptic stricture includes intralesional steroid injections, peroral esophageal dilatations, and surgery (Edmundowicz 1999; O'Connor and Richter 1999). Steroid injections into the site of stricture have been controversial, but may reduce inflammation and delay or possibly prevent fibrosis; however, patients having this form of therapy often require dilatations or even anti-reflux surgery.

Esophageal dilatation is usually the initial method of choice for treatment of benign strictures and has proven safe and effective in relieving associated dysphagia (Edmundowicz 1999; O'Connor and Richter 1999; Pereira-Lima et al. 1999; Scolapio et al. 1999). Esophageal dilatation involves repetitive passage of increasingly larger dilators across a stricture, usually on a frequent basis, until a lumen size of 14–15 mm or larger is obtained. The three main types of dilators used in the United States are: 1) mercury-filled rubber types (Maloney or Hurst); 2) wire-guided Savary-Gilliard dilators (Fig. 5.4.11); and 3) balloon dilators that can be passed endoscopically or over a guide wire. These procedures may be performed using fluoroscopic guidance, but may be done in an outpatient setting, or performed by the patient at home. Esophageal perforation is the most important complication of stricture dilation, although a variety of lesser problems, such as placement in the trachea or curling in the esophagus, may occur (Edmundowicz 1999; O'Connor and Richter 1999; Saeed et al. 1997). The esophageal perforation rate with the more modern dilators is extremely low, and is about 0.25%

Fig. 5.4.10. Focal narrowing of the lower esophagus (*arrows*) due to peptic stricture

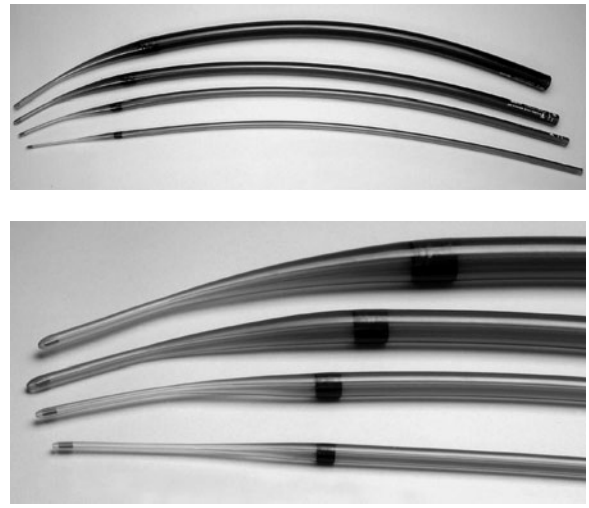


Fig. 5.4.11a,b. **a** Savary-Gilliard esophageal dilators (Wilson-Cook Medical, Winston-Salem, NC) of various sizes. **b** Tapered tips of the same dilators. Sizes at the level of the dark rings are 20 mm (60F), 15 mm (45F), 11 mm (33F), and 8 mm (24F) from the top to bottom, respectively

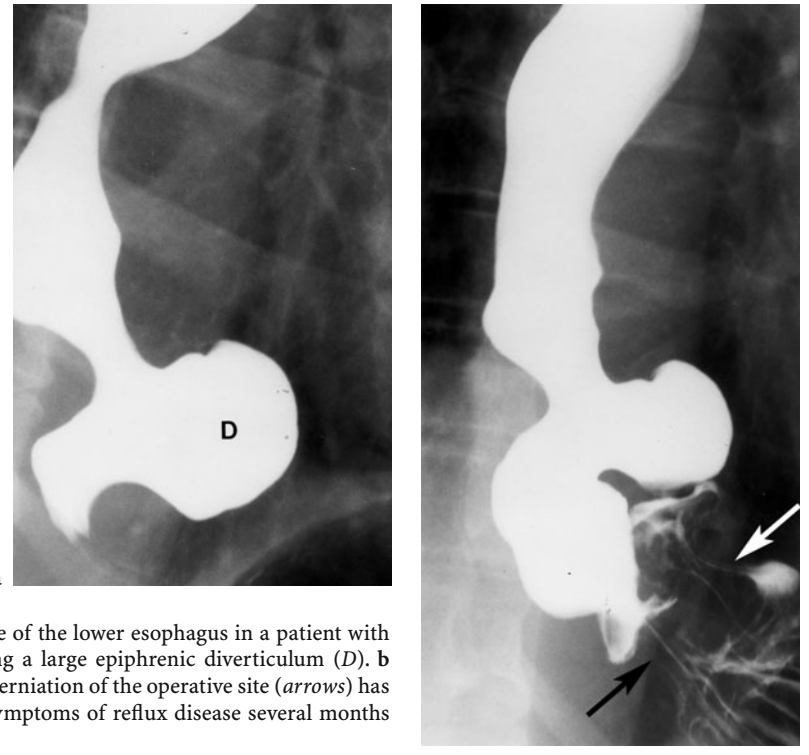
per procedure; however, the risk is higher in more complex and narrower strictures (O'Connor and Richter 1999). Radiographic evaluation of patients following stricture dilation with suspected esophageal injury is similar to evaluating any individual with suspected esophageal perforation.

Surgical therapy for peptic stricture is indicated in those who have failed repeated esophageal dilatations or are not suitable for aggressive medical therapy (Edmundowicz 1999; O'Connor and Richter 1999). Anti-reflux operations available to patients with reflux disease and often refractory complications have evolved in recent years from open thoracic or abdominal procedures to those performed using laparoscopic techniques (Hatfield and Shapir 1985; Klingler et al. 1999; Luostarinen 1993; Skucas et al. 1976). The standard open Nissen fundoplication was the most commonly used procedure, and whether partial or complete fundic wraps were used depended on factors such as the effectiveness of esophageal peristalsis and potential avoidance of post-operative dysphagia or 'gas-bloat' syndrome. These procedures have been modified for laparoscopic use; however, post-operative complications, both acute and late in origin, can occur which may warrant radiographic evaluation (Fig. 5.4.12) (Klingler et al. 1999).

5.4.7 Esophageal Malignancy

Squamous cell carcinoma and adenocarcinoma are the most common primary esophageal malignancies (Chen et al. 1999; Jenkins and Friedman 1999; Wolfman et al. 1994). Early diagnosis of esophageal carcinoma portends a better prognosis because the size of the lesion and extent of invasion into the esophageal wall are related; however, in the absence of effective screening programs, esophageal malignancy is usually diagnosed as a more advanced tumor, which has invaded adjacent organs and is associated with a poor survival (Fig. 5.4.13). Adenocarcinoma has increased dramatically in prevalence; this malignancy typically originates in the lower esophagus and may arise from the esophageal glands, heterotopic gastric mucosal, or metaplastic columnar epithelium, so-called Barrett's carcinoma (Cameron et al. 1995; Jenkins and Friedman 1999; Sampliner 1998). Indeed, the most common origin is believed to be dysplastic Barrett's epithelium. Surgery for esophageal malignancy may be performed for curative intent, but endoscopic procedures and the use of a variety of endoluminal stents can be used to manage these patients.

Malignant obstruction of the esophagus and palliation of dysphagia or associated complications (e.g.



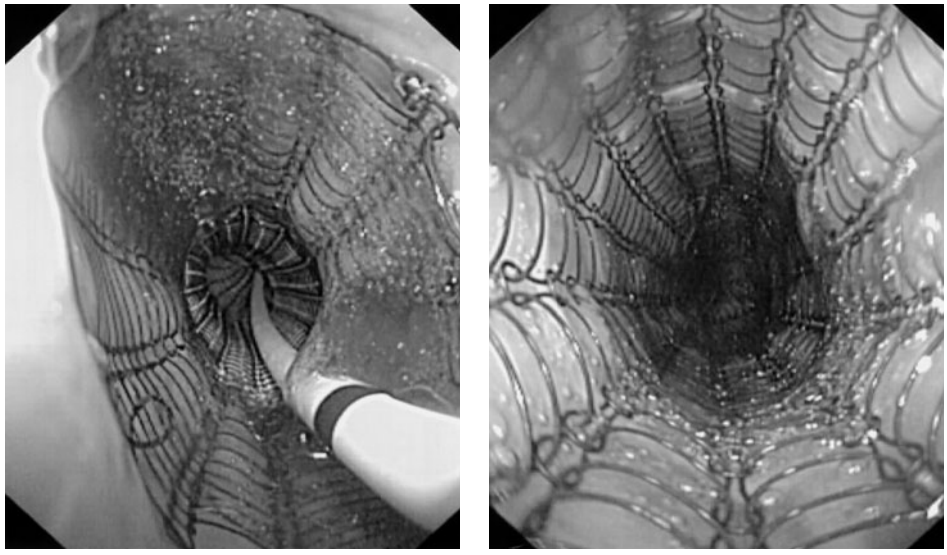
a b

Fig. 5.4.12a,b. a Pre-operative appearance of the lower esophagus in a patient with gastro-esophageal reflux disease showing a large epiphrenic diverticulum (*D*). **b** Following laparoscopic fundoplication, herniation of the operative site (*arrows*) has occurred. Patient developed recurrent symptoms of reflux disease several months after the procedure

Fig. 5.4.13 Advanced, annular squamous cell carcinoma of the lower esophagus

mediastinal sinus tract or airway fistula) are commonly treated with endoscopic methods (Edmundowicz 1999). Mucosal ablation or resection techniques for possible curative treatment of early esophageal malignancy or management of Barrett's esophagus were discussed previously. A tissue diagnosis is important initially because esophageal stricture or obstruction may be due to a benign or a malignant cause and early treatment may be similar. Tumor ablation combined with serial dilatations can destroy and reduce malignant tissue bulk and also increase the luminal diameter of the esophagus to relieve dysphagia and improve nutrition of the patient. If the various tumor ablation techniques and standard dilatations are not successful or need to be repeated frequently, endoscopic stent placement offers another option.

Endoscopic stent placement for malignant obstruction of the esophagus has evolved in recent years from the use of plastic stents to the deployment of expandable metallic stents (Adam et al. 1998; Bethge and Vakil 2001; Cwikiel et al. 1998; Ell and May 1997; Knyrim et al. 1993; Mosca et al. 2000; Vakil et al. 2001). Large diameter plastic stents were successful in relieving dysphagia in selected patients, but have been replaced by the metallic stents; complications included migration, tumor overgrowth, and obstruction, which were evaluated with radiographic filming and contrast examinations. Expandable metallic stents provide a newer approach to palliative treatment of malignant strictures that offers several advantages (Fig. 5.4.14). The stent introducers are of a smaller diameter (some pass through the endoscopic channels), which limits the need for extensive dilatation of the area of malignant narrowing, thus avoiding potential complications, such as perforation of the esophagus (Fig. 5.4.15). The expandable stents are also designed to slowly create radial forces that enlarge the luminal diameter until the



a **b**

Fig. 5-14a,b. a Endoscopic image of partial deployment of a metallic esophageal stent. **b** Same stent after full deployment

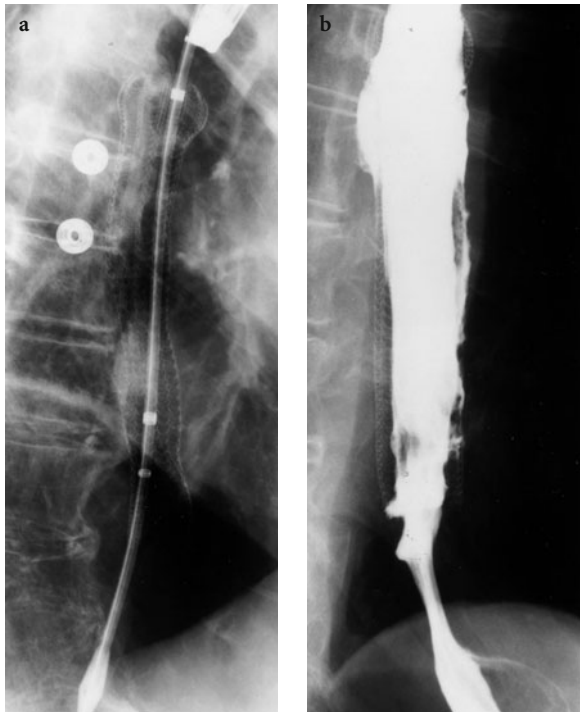


Fig. 5.4.15a,b. a Radiographic appearance of partial deployment of an esophageal metallic stent for palliative treatment of esophageal carcinoma. **b** Water-soluble contrast examination of the esophagus the next day shows full deployment of the stent and patency of the lumen without complications, such as perforation

stent is expanded fully. Coated stents are available which help to prevent tumor in-growth through the metallic mesh and permit the closure of sinus tracts, fistulas, and perforations. Disadvantages of metallic stents include their cost, difficulties with migration, and the continued problem of tumor growth and occlusion. As with plastic stents, radiographic imaging is helpful in evaluation of the position of the stents in the esophagus and associated complications (Fig. 5.4.16) (Gollub et al. 1997; Morgan et al. 1997; Nevitt et al. 1996).

5.4.8 Conclusion

In conclusion, management of esophageal diseases in the past consisted primarily of diagnosis and possibly surgical or radiation treatment. Major advancements in esophageal intervention have occurred in recent years, especially with the use of endoscopic techniques. The endoscopist typically performs many of these interventional procedures, but imaging may be require before, during, or after the endoscopic intervention is performed. In this chapter, we have discussed a variety of esophageal problems amenable to endoscopic intervention and reviewed the role of imaging in assisting in the management of these disorders and their potential complications.

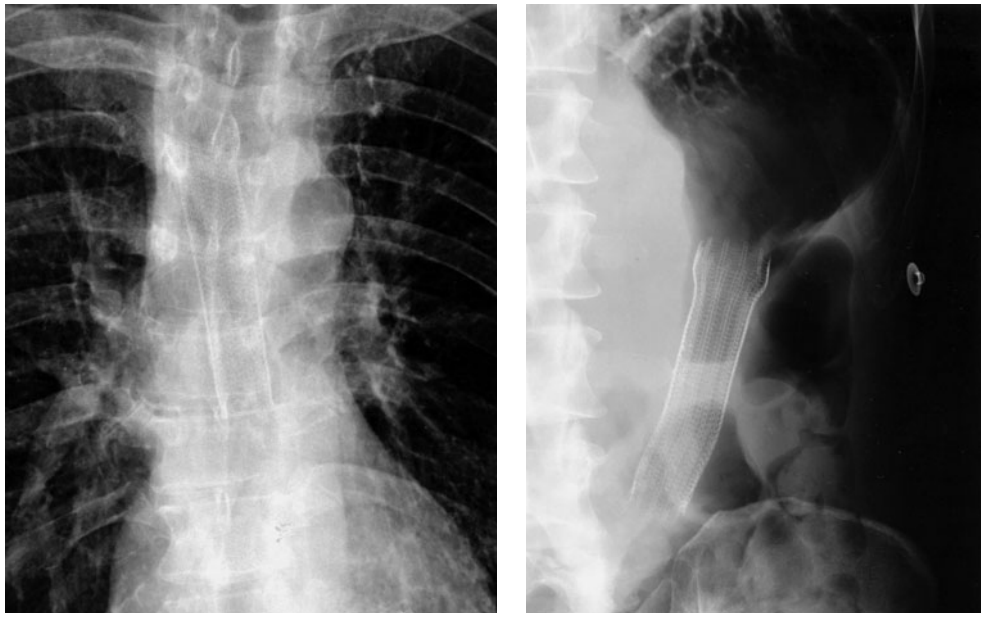


Fig. 5.4.16a,b. a Fully deployed metallic stent in the mid-esophagus at the level of the aortic arch and carina on a plain chest radiograph. **b** Patient developed dysphagia and the stent was no longer seen in its previous location on the chest film. An abdominal radiograph showed that the stent had dislodged and migrated into the stomach

References

Ackroyd R, Watson DI, Devitt PG, Jamieson GG (2001) Laparoscopic cardiomyotomy and anterior partial fundoplication for achalasia. Surg Endosc 15:683–686

Adam A, Morgan R, Ellul J, Mason RC (1998) A new design of the esophageal Wallstent endoprosthesis resistant to distal migration. AJR 170:1477–1481

Agha FP (1984) The esophagus after endoscopic injection sclerotherapy: acute and chronic changes. Radiology 153:37–42

Armstrong D (1994) Reflux disease and Barrett's esophagus. Endoscopy 26:9–19

Bass NM, Somberg KA (1998). Portal hypertension and gastrointestinal bleeding In: Feldman M, Scharschmidt BF, Sleisenger MH (eds) Gastrointestinal and liver disease, 6th edn. W.B. Saunders, Philadelphia, pp 1284–1309

Bethge N, Vakil N (2001) A prospective trial of a new self-expanding plastic stent for malignant esophageal obstruction. Am J Gastroenterol 96:1350–1354

Buttar NS, Wang KK, Sebo TJ, Riehle DM, Krishnadath KK, Lutzke LS, Anderson MA, Petterson TM, Burgart LJ (2001) Extent of high-grade dysplasia in Barrett's esophagus correlates with risk of adenocarcinoma. Gastroenterology 120: 1630–1639

Cade R (2000) Heller's myotomy: thoracoscopic or laparoscopic? Dis Esophagus 13:279–281

Cameron AJ, Lomboy CT, Pera M, Carpenter HA (1995) Adenocarcinoma of the esophagogastric junction and Barrett's esophagus. Gastroenterology 109:1541–1546

Chen LQ, Hu CY, Ghadirian P, Duranceau A (1999) Early detection of esophageal squamous cell carcinoma and its effects on therapy: an overview. Dis Esophagus 12:161–167

Colon V, Grade A, Pulliam G, Johnson C, Fass R (1999) Effect of doses of glucagon used to treat food impaction on esophageal motor function of normal subjects. Dysphagia 14:27–30

Cwikiel W, Tranberg KG, Cwikiel M, Lillo-Gil R (1998) Malignant dysphagia: palliation with esophageal stents–long-term results in 100 patients. Radiology 207:513–518

Eckardt VF, Kanzler G, Westermeler T (1997) Complications and their impact after pneumatic dilation for achalasia: prospective long-term follow-up study. Gastrointest Endosc 45:349–353

Edmundowicz SA (1999) Endoscopy. In: Castell DO, Richter JE (eds) The esophagus, 3rd edn. Lippincott, Williams, & Wilkins, Philadelphia, pp 89–99

Ell C, May A (1997) Self-expanding metal stents for palliation of stenosing tumors of the esophagus and cardia: a critical review. Endoscopy 29:392–398

Ell C, May A, Gossner L, Pech O, Günter E, Mayer G, Henrich R, Vieth M, Müller H, Seitz G, Stolte M (2000) Endoscopic mucosal resection of early cancer and high-grade dysplasia in Barrett's esophagus. Gastroenterology 118:670–677

Glick SN (1994) Barium studies in patients with Barrett's esophagus: importance of focal areas of esophageal deformity. AJR 163:65–67

Gollub MJ, Gerdes H, Bains MS (1997) Radiographic appearances of esophageal stents. Radiographics 17:1169–1182

Hatfield M, Shapir J (1985) The radiologic manifestations of failed antireflux operations. AJR 144:1209–1214

Imperiale TF, O'Connor B, Vaezi MF, Richter JE (2000) A cost-minimization analysis of alternative treatment strategies for achalasia. Am J Gastroenterol 95:2737–2745

Jenkins TD, Friedman LS (1999) Adenocarcinoma of the esophagogastric junction. Dig Dis 17:153–162

Karasick S, Lev-Toaff AS (1995) Esophageal strictures: findings on barium radiographs. AJR 165:561–565

Kaszar-Seibert DJ, Korn WT, Bindman DJ, Shortsleeve MJ (1990) Treatment of acute esophageal food impaction with a combination of glucagon, effervescent agent, and water. AJR 154:533–534

Katilius M, Velanovich V (2001) Heller myotomy for achalasia: quality of life comparison of laparoscopic and open approaches. JSLS 5:227–231

Katz PO, Gilbert J, Castell DO (1998) Pneumatic dilatation is effective long-term treatment for achalasia. Dig Dis Sci 43: 1973–1977

Klingler PJ, Bammer T, Wetscher GJ, Glaser KS, Seelig MH, Floch NR, Branton SA, Hinder RA (1999) Minimally invasive surgical techniques for the treatment of gastroesophageal reflux disease. Dig Dis 17:23–36

Knyrim K, Wagner HJ, Bethge N, Keymling M, Vakil N (1993) A controlled trial of an expansile metal stent for palliation of esophageal obstruction due to inoperable cancer. N Engl J Med 329:1302–1307

Kochhar R, Goenka MK, Mehta SK (1992) Esophageal strictures following endoscopic variceal sclerotherapy. Dig Dis Sci 37:347–352

Kolbasnik J, Waterfall WE, Fachnie B, Chen Y, Tougas G (1999) Long-term efficacy of Botulinum toxin in classical achalasia: a prospective study. Am J Gastroenterol 94:3434–3439

Kumar V, Shimi SM, Cuschieri A (1998) Does laparoscopic cardiomyotomy require an antireflux procedure? Endoscopy 30:8–11

Levine MS, Herman JB, Furth EE (1995) Barrett's esophagus and esophageal adenocarcinoma: the scope of the problem. Abdom Imaging 20:291–298

Longstreth GF, Longstreth KJ, Yao JF (2001) Esophageal food impaction: epidemiology and therapy–a retrospective, observational study. Gastrointest Endosc 53:193–198

Luostarinen M (1993) Nissen fundoplication for reflux esophagitis–long-term clinical and endoscopic results in 109 of 127 consecutive patients. Ann Surg 217:329–337

Macpherson RI, Hill JG, Othersen HB, Tagge EP, Smith CD (1996) Esophageal foreign bodies in children: diagnosis, treatment, and complications. AJR 166:919–924

Maglinte DDT, Chernish SM, Kelvin FM, Lappas JC (1995) Pharmacoradiologic disimpaction of esophageal foreign bodies: review and recommendation. Emergency Radiol 2:151–157

Mamel JJ, Weiss D, Pouagare M, Nord HJ (1995) Endoscopic suction removal of food boluses from the upper gastrointestinal tract using Stiegmann-Goff friction-fit adaptor: an improved method for removal of food impactions. Gastrointest Endosc 41:593–596

Metman EH, Lagasse JP, d'Alteroche L, Picon L, Scotto B, Barbieux JP (1999) Risk factors for immediate complications after progressive pneumatic dilation for achalasia. Am J Gastroenterol 94:1179–1185

Molina EG, Stollman N, Grauer L, Reiner DK, Barkin JS (1995) Conservative management of esophageal nontransmural tears after pneumatic dilation for achalasia. Am J Gastroenterol 91:15–18

Morgan RA, Ellul JPM, Denton ERE, Glynos M, Mason RC,

Adam A (1997) Malignant esophageal fistulas and perforations: management with plastic-covered metallic endoprostheses. Radiology 204:527–532

Mosca F, Stracqualursi A, Portale TR, Consoli A, Latteri S (2000) Palliative treatment of malignant esophageal stenosis: the role of self-expanding stent endoscopic implantation. Dis Esophagus 13:301–304

Nevitt AW, Kozarek RA, Kidd R (1996) Expandable esophageal prostheses: recognition, insertion techniques, and positioning. AJR 167:1009–1013

O'Connor JB, Richer JE (1999) Esophageal strictures. In: Castell DO, Richter JE (eds) The esophagus, 3rd edn. Lippincott, Williams, & Wilkins, Philadelphia, pp 473–483

Ott DJ (1994) Gastroesophageal reflux disease. Radiol Clinics North Am 32:1147–1166

Ott DJ, Chen MYM (1998) What is the value of the radiological diameter of the esophagogastric junction following dilation? In: Giuli R, Galmiche JP, Jamieson GG, Scarpignato C (eds) The esophagogastric junction. John Libbey, Paris, pp 1356–1359

Ott DJ, Richter JE, Wu WC, Chen YM, Castell DO, Gelfand DW (1987) Radiographic evaluation of esophagus immediately after pneumatic dilatation for achalasia. Dig Dis Sci 32: 962–967

Ott DJ, Donati D, Wu WC, Chen MYM, Gelfand DW (1991) Radiographic evaluation of achalasia immediately after pneumatic dilatation with the Rigiflex dilator. Gastrointest Radiol 16:279–282

Pereira-Lima JC, Ramires RP, Zamin I Jr, Cassal AP, Marroni CA, Mattos AA (1999) Endoscopic dilation of benign esophageal strictures: report on 1043 procedures. Am J Gastroenterol 94:1497–1501

Prakash C, Freedland KE, Chan MF, Clouse RE (1999) Botulinum toxin injections for achalasia symptoms can approximate the short term efficacy of a single pneumatic dilation: a survival analysis approach. Am J Gastroenterol 94:328–333

Rice BT, Spiegel PK, Dombrowski PJ (1983) Acute esophageal food impaction treated by gas-forming agents. Radiology 146:299–301

Saeed ZA, Michaletz PA, Feiner SD, Woods KL, Graham DY (1990) A new endoscopic method for managing food impaction in the esophagus. Endoscopy 22:226–228

Saeed ZA, Ramirez FC, Hepps KS, Cole RA, Schneider FE, Ferro PS, Graham DY (1997) An objective end point for dilation improves outcome of peptic esophageal strictures: a prospective randomized trial. Gastrointest Endosc 45: 354–359

Sampliner RE (1998) Practice guidelines on the diagnosis, surveillance, and therapy of Barrett's esophagus. Am J Gastroenterol 93:1028–1032

Schnell TG, Sontag SJ, Chejfec G, Aranha G, Metz A, O'Connell S, Seidel UJ, Sonnenberg A (2001) Long-term nonsurgical management of Barrett's esophagus with high-grade dysplasia. Gastroenterology 2001:1607–1619

Scolapio JS, Pasha TM, Gostout CJ, Mahoney DW, Zinsmeister AR, Ott BJ, Lindor KD (1999) A randomized prospective study comparing rigid to balloon dilators for benign esophageal strictures and rings. Gastrointest Endosc 50:13–17

Sharara AI, Rockey DC (2001) Gastroesophageal variceal bleeding. N Engl J Med 345:669–681

Sharma P, Topalovski M, Mayo MS, Weston AP (2001) Methylene blue chromoendoscopy for detection of short-segment Barrett's esophagus. GI Endosc 54:289–293

Skucas J, Mangla JC, Adams JT, Cutcliff W (1976) An evaluation of the Nissen fundoplication. Radiology 118:539–543

Soni A, Sampliner RE, Sonnenberg A (2000) Screening for high-grade dysplasia in gastroesophageal reflux disease: is it cost-effective. Am J Gastroenterol 95:2086–2093

Subramanyam K, Patterson M (1986) Chronic esophageal ulceration after endoscopic sclerotherapy. J Clin Gastroenterol 8:58–60

Tihansky DP, Reilly JJ, Schade RR, Van Thiel DH (1984) The esophagus after injection sclerotherapy of varices: immediate postoperative changes. Radiology 153:43–47

Trenkner SW, Maglinte DDT, Lehman GA, Chernish SM, Miller RE, Johnson CW (1983) Esophageal food impaction: treatment with glucagons. Radiology 149:401–403

Vaezi MF, Richter JE (1999) Diagnosis and management of achalasia. Am J Gastroenterol 94:3406–3412

Vaezi MF, Baker ME, Richter RE (1999) Assessment of esophageal emptying post-pneumatic dilation: use of the timed barium esophagram. Am J Gastroenterol 94:1802–1807

Vakil N, Morris AI, Marcon N, Segalin A, Peracchia A, Bethge N, Zuccaro G, Bosco JJ, Jones WF (2001) A prospective, randomized, controlled trial of covered expandable metal stents in the palliation of malignant esophageal obstruction at the gastroesophageal junction. Am J Gastroenterol 96:1791–1796

van den Boogert J, van Hillegersberg R, Siersema PD, de Bruin RWF, Tilanus HW (1999) Endoscopic ablation therapy for Barrett's esophagus with high-grade dysplasia: a review. Am J Gastroenterol 94:1153–1160

Weinstock LB, Shatz BA, Thyssen EP (1999) Esophageal food bolus obstruction: evaluation of extraction and modified push techniques in 75 cases. Endoscopy 31:421–425

Wolfman NT, Scharling ES, Chen MYM (1994) Esophageal squamous carcinoma. Radiol Clin North Am 32: 1183–1201

Yeaton P, Peura DA (1999) Foreign bodies. In: Castell DO, Richter JE (eds) The esophagus, 3rd edn. Lippincott, Williams, & Wilkins, Philadelphia, pp 335–348

6 Endoscopy of the Pharynx and Esophagus

Doris-Maria Denk and Rainer Schöfl

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6.1 Introduction

The pharynx and esophagus belong to the upper digestive tract; their morphology and function enable normal swallowing. Dysphagia is one of the main symptoms in patients with pharyngeal and esophageal diseases. Diagnostics need to consider the entire swallowing chain from the oral cavity to the stomach. However, there is a close relationship between diseases of the pharynx, larynx and esophagus: gastrointestinal disorders, e.g. reflux disease, may show extraesophageal manifestations in the pharynx and larynx, and in case of tumours in the upper aerodigestive tract or esophagus, there may occur additional simultaneous tumours urging for early diagnosis.

For diagnostic evaluation, endoscopy has emerged as the "first line" examination. Technical progress has improved the quality of endoscopic imaging and enabled video documentation in clinical routine. In addition to the endoscopic visualization of the aerodigestive tract, biopsies allow histological diagnosis, and therapeutic manipulations can be performed. Together with radiology, endoscopy has become indispensable in the management of diseases of the pharynx and esophagus. A main focus of diagnostic interest is the differential diagnosis between structural diseases and functional disorders. For therapeutic decision-making, it is of utmost importance to prove or exclude malignancy and aspiration.

Patients with pharyngeal and esophageal diseases will often cross specialty lines. About 80% of patients with esophageal disorders present first to an otolaryngologist because they suffer from head and neck symptoms, like dysphagia, cough or globus sensation. Whereas otolaryngology deals with laryngo-pharyngeal disorders, the management of esophageal diseases falls more appropriately within the realm of gastroenterology, which is competent for the whole intestine, as well as thoracic surgery. This interdisciplinary approach is reflected by the two authors of this chapter.

Endoscopy and radiology are not the only instrumental methods in the diagnostic armamentarium. In addition, depending on the patient's symptoms and endoscopic or radiologic findings, further examinations may be needed for conclusive analysis of the pharynx and esophagus. For example, manometry, manofluorography, pH-metry, scintigraphy for

D.-M. Denk, MD
Professor, Department of Otorhinolaryngology, Section of Phoniatrics, Vienna Medical School, University of Vienna, Währinger Gürtel 18–20, 1090 Vienna, Austria
R. Schöfl, MD
4th Department of Internal Medicine, Hospital of the Elisabethinen, Linz and Department of Internal Medicine IV, Section of Gastroenterology and Hepatology, University of Vienna, 1090 Vienna, Austria

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quantification of the pharyngo-esophageal transport, or electromyography provide valuable information.

It is the goal of this chapter to describe the endoscopic examination of the upper aerodigestive tract and examples of typical findings. Advantages and limitations of endoscopy are demonstrated. Moreover, the role of endoscopy within the diagnostic work-up of dysphagic patients and future aspects will be discussed.

6.2 Endoscopy of the Pharynx and Larynx

A critical area for deglutition without aspiration is crossing of the airway and digestive tract, which is localized in the hypopharynx. Apart from deglutition, pharyngeal structures belong to the vocal tract, which is responsible for articulation and resonance. Therefore, the pharynx and larynx as part of the upper aerodigestive tract have to be evaluated in context by the otolaryngologist. With the mirror examination, the ability to adequately visualize the pharynx and larynx may be limited. Therefore, endoscopy has become a standard examination in clinical routine.

Indications for endoscopy of the pharynx and larynx are patients presenting with symptoms of respiratory and swallowing diseases. Symptoms are dysphagia, aspiration, regurgitation, odynophagia and dysphonia (hoarseness). Before endoscopy is carried out, a history has to be taken and a mirror examination of ears, nose, mouth, pharynx and larynx is performed.

The indirect endoscopy of the pharynx and larynx views their inner surface via optical instruments. It can be performed transorally with rigid endoscopes (telescopes) and transnasally with flexible endoscopes. Video documentation should be obtained whenever possible.

6.2.1 Indirect Rigid Endoscopy of the Hypopharynx and Larynx

Rigid 70° and 90° degree telescopes (Fig. 6.1) are used to evaluate the hypopharynx and larynx indirectly. They provide a magnified view in high resolution, allow videotaping for documentation and can be used in association with stroboscopy to evaluate vocal fold vibrations. In addition, indirect rigid endoscopy enables the performance of office-based laryngeal surgical procedures under topical anaesthesia (biopsies, indirect phonosurgery for voice improvement).

Fig. 6.1. Rigid telescope and flexible rhinolaryngoscope

For endoscopy, the patient sits in an upright position, with his tongue protruded, and the examiner gently inserts the objective end of the telescope posteriorly over the base of the tongue until the hypopharynx and larynx are seen. The unphysiological patient condition allows only the examination of "hi"-phonation, but not the direct observation of articulation and swallowing. In cases of gag reflex, local anaesthesia can facilitate the examination. The observer examines morphology (e.g. signs of inflammation, tumour) and function (vocal fold mobility, pooling/aspiration of saliva).

6.2.2 Flexible Endoscopy of the Pharynx and Larynx

Transnasal flexible nasopharyngolaryngoscopy is a common procedure among otorhinolaryngologists for assessing nasal, velopharyngeal and laryngeal pathology. Moreover, it is used for the evaluation of oropharyngeal swallowing and as a treatment tool in biofeedback therapy of voice and swallowing disorders.

The flexible rhinolaryngoscope consists of an objective lens at the distal end of the insertion portion of the endoscope. The diameter of the insertion tube of the scope is kept as small as possible (3–4 mm). A xenon or halogen light source is used. With a chip camera the image is viewed on a monitor and recorded on a video recorder.

The flexible nasopharyngolaryngoscope is inserted through the nasal passage, a topical anaesthesia (e.g. anaesthetic and decongestant spray) can be applied

before. The examination starts with the visualization of the nasal fossa, the epipharynx and velum. The velopharyngeal competence and closure is tested. Then, the endoscope is passed down to just above the epiglottis where the larynx and hypopharynx can be seen (panoramic view). Tongue base, position and morphology of the epiglottis, configuration of the posterior pharyngeal wall, vallecular spaces, piriform sinuses, arytenoids and vocal folds are investigated. After that, the scope is moved further down just above the vocal folds for detailed inspection (larynx view). Signs of inflammation, mucosal abnormalities, mass lesions and vocal fold mobility can easily be observed. If local anaesthesia was administered to reduce the cough reflex, the glottic level can be passed for inspection of the subglottic region and trachea.

Flexible endoscopy is also appropriate for patients who cannot be examined by mirror or rigid endoscopy because of a strong gag reflex and for patients who are not able to cooperate, e.g. paediatric or emergency patients. The main advantage is that the flexible endoscope can be left in position during phonation, articulation and swallowing, therefore it is used for evaluation of functions. As a limitation it has to be pointed out that the image quality of flexible endoscopy does not reach that of rigid endoscopy. Whenever videostroboscopy is performed, the method of choice is rigid endoscopy.

Our experience with more than 4000 endoscopies shows that flexible endoscopy is a procedure without major complications. However, vasovagal reaction, laryngospasm or epistaxis are described in the literature. Therefore, all measurements for managing such complications must be available.

6.2.3 Examples of Typical Findings

Normal Hypopharynx and Larynx. The base of the tongue, posterior pharyngeal wall, piriform sinus and supraglottis are covered by intact epithelium, the vocal folds present in white colour due to the non-keratinizing squamous epithelium. Both vocal folds move symmetrically between the respiration and phonation position. No pooling of saliva or food is seen.

Hypopharynx Carcinoma. Foreign tissue and perhaps ulceration is seen in the hypopharynx (posterior pharyngeal wall, piriform sinus or postcricoid region). Due to tumour infiltration, vocal fold motility may be disturbed. Figure 6.2 shows an exophytic tumour

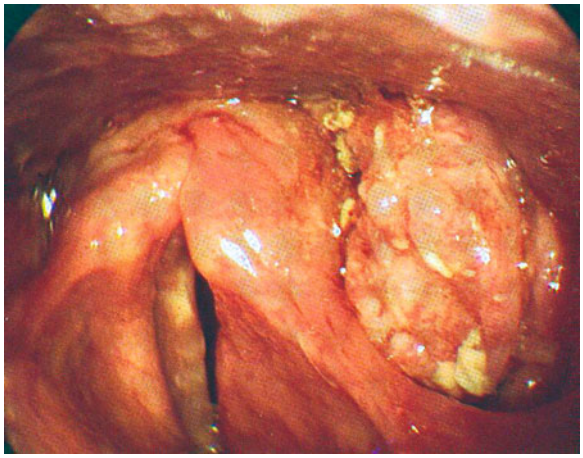


Fig. 6.2. Hypopharynx carcinoma tumour mass in the left pyriform sinus (indirect rigid hypopharyngolaryngoscopy [From Becker et al. (1983)]

mass in the left piriform sinus, reaching the postcricoid wall. Due to tumour infiltration the left vocal fold is paralysed. The patient's symptoms included dysphonia, dyspnea and long-lasting slowly progressive dysphagia.

Reflux Laryngitis. Gastroesophageal/pharyngeal reflux disease can lead to a laryngitis which is not always limited to the posterior larynx. Possible morphological findings are reddening of the arytenoids, hypertrophy in the posterior commissure, contact granuloma of the vocal process or glottic/subglottic stenosis.

Reinke's Oedema. This type of chronic laryngitis frequently occurs in smokers, in patients with vocal abuse and in endocrinological dysfunctions (e.g. menopause, hypothyroidism). It presents with oedematous, thickened vocal folds and vasectasias (Fig 6.3). The voice typically sounds low and frequently hoarse.

Leukoplakia of the Vocal Folds. The epithelium is covered by a white coat, and distinction from malignancy can only be made histologically. Stroboscopy helps to judge whether the process is infiltrating or not, but cannot replace histology. If the vocal fold does not vibrate in stroboscopy, an infiltrating process is present, and urgent microlaryngoscopy with biopsy for histological examination is indicated.

Unilateral Vocal Fold Paralysis. The paralysed vocal fold is in fixed position (median, paramedian, intermediate or lateral), the arytenoid may be dislocated anteriorly. During phonation, depending on the posi-

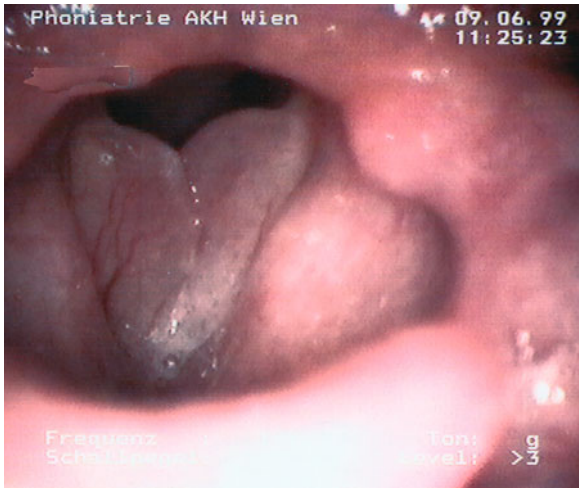


Fig. 6.3. Reinke's oedema (rigid hypopharyngolaryngoscopy)

Fig. 6.4. Vocal fold paresis, left side (flexible hypopharyngolaryngoscopy)

tion of the vocal fold, glottic closure is incomplete. Stroboscopy may be of some prognostic value: the presence of the mucosal wave is a good prognostic sign. Figure 6.4 shows a left-sided vocal fold paresis in paramedian position.

6.2.4 Flexible Endoscopic Evaluation of Swallowing

The flexible endoscopic evaluation of swallowing (FEES), also called videoendoscopic swallowing study (VESS), was introduced by Langmore et al. (1988) and Bastian (1991). This dynamic diagnostic method allows an evaluation of the oropharyngeal swallow and has become a routine otorhinolaryngological/phoniatric procedure. It aims at defining the individual swallowing profile of the patient examined, which enables an adequate treatment planning and recommendation for feeding. FEES is regarded to be more than a screening procedure and does not only identify dysphagia and aspiration, but reveals the pathophysiology of the swallowing disorder.

According to the modified barium swallow, FEES is performed as a tailored examination (Bigenzahn and Denk 1999; Schröter-Morasch 1999; Schröter-Morasch et al. 1999; Langmore 2001): coloured food in different consistencies is used depending on the history and clinical findings. Suction must be available in case of aspiration. Before endoscopy, the patient has to be observed in the clinical examination, and neurological symptoms or disorders of speech, language or voice have to be noted.

The patient is in an upright position with the head slightly down to facilitate swallowing function. Generally, no local anaesthetic spray is used in order not to impair pharyngolaryngeal sensibility. If needed, only cotton balls with local anaesthetic and decongestant are positioned into the nose before endoscopy. The flexible rhinopharyngolaryngoscope is introduced transnasally into the oro- and hypopharynx and is left in place during deglutition. Videotaping allows an analysis in slow motion and discussion of the findings in the interdisciplinary management team.

The endoscopic examination consists of two parts: non-swallowing and swallowing assessment. In the "non-swallowing assessment", anatomy and function are investigated. The mobility of the vocal folds, the occurrence of hyperkinetic movements, pooling/aspiration of saliva, cough reflex (elicited by gently touching the glottis with the tip of the endoscope) and the possibility of intentional (voluntary) throat clearing are tested. Velum, pharynx and larynx are observed not only during respiration, but also during phonation, breathhold manoeuvres, throat clearing and coughing to test the intentional and reflexive mobility. The second part of the procedure comprises the swallowing assessment, i.e. "dry swallow" with saliva and "food swallows" with measured quantities of food and liquid of different consistencies, dyed with blue food colouring. The endoscope is positioned in the panoramic view above the tip of the epiglottis. In tracheostomized patients, endoscopy via the tracheostoma is performed in addition.

Swallowing function is evaluated with regard to saliva pooling, triggering of the swallowing reflex,

leaking, penetration, retention, aspiration, cough reflex, and regurgitation. Regurgitation leads to suspicion of a hypopharyngeal/esophageal stenosis or Zenker's diverticulum. Aspiration before and after the swallow can be viewed, whereas aspiration during the swallow cannot be seen directly. Also the amount of aspiration cannot be judged securely (only in patients without cough reflex or tracheostomy). The ability to effectively clear the throat of retention and aspirated material is tested. Finally, compensatory postures, swallowing techniques and different food consistencies are evaluated for establishing an individually tailored treatment program. Moreover, flexible endoscopy has proved to be a treatment tool for visual biofeedback training in functional swallowing therapy (Denk and Kaider 1997). The combination with other diagnostic procedures may be useful. Commercially available "workstations" eventually comprise sonography, electromyography or other diagnostic methods in addition. Figure 6.5 shows a static image of aspiration. Coloured liquid is pouring down the subglottic region into the trachea.

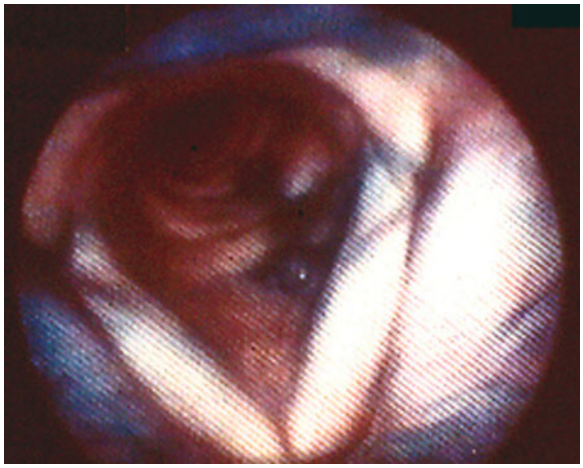


Fig. 6.5. Aspiration (flexible hypopharyngolaryngoscopy) [From Bigenzahn and Denk (1999)]

6.2.4.1 Advantages and Limitations

The following *limitations* of FEES have to be taken into account:

- There is no direct visualization of the bolus on its entire way from mouth to stomach as offered by videofluoroscopy.
- Laryngeal closure (because of epiglottic tilting) and aspiration during the swallow cannot be examined directly. The view during the swallow is obscured because pharyngeal mucosa and the bolus touches the tip of the endoscope.
- Larynx/hyoid elevation and upper esophageal sphincter function are not shown. Diseases of the pharyngoesophageal segment and esophagus can only be indirectly supposed in the case of pharyngeal residue and/or pharyngeal regurgitation.
- Routinely, esophagoscopy is not part of the examination. Some authors propose to use a longer flexible endoscope to routinely evaluate the esophagus during FEES (Herrmann 1998).

The influence of the endoscope as a foreign body during swallowing has not yet been evaluated exactly.

On the contrary, there are many *advantages* of FEES:

- The direct visualization of the upper-aerodigestive tract reveals even subtle morphological or functional findings.
- It is a non-invasive procedure without any radiation exposure, repeatable as often as necessary and available also as a bedside examination.
- Regular food, not barium, is used.

6.2.4.2 Comparison of FEES and Videofluoroscopy

The only methods for visualization of aspiration are videofluoroscopy (VFS) and FEES. VFS visualizes the bolus on its all the way from the oral cavity to the stomach and was the first instrumental procedure for the assessment of dysphagia (Logemann 1993, 1998; Jones and Donner 1991; Ekberg and Olsson 1997). FEES directly shows the upper aerodigestive tract. Especially with regard to cost effectiveness, the question arises as to which method is best for the evaluation of dysphagia. Literature and own studies show that these dynamic methods are not alternative, but complementary procedures (Schima and Denk 1998). Both are valuable, each procedure has its place in the clinical setting. Together they represent the gold standard in dysphagia evaluation.

Comparing the findings of FEES and VFS, there is excellent agreement with regard to aspiration and retention. Due to the limitations of FEES mentioned above, VFS remains indispensable for the evaluation of the complete upper digestive tract in one examination. FEES is the method of choice for the first-line examination, follow-up examinations, for unfit patients in the intensive care unit and for the evaluation of compensatory manoeuvres.

6.2.4.3
Flexible Endoscopic Evaluation of Swallowing with Sensory Testing

Flexible endoscopic evaluation of swallowing with sensory testing (FEESST) is the combination of FEES with laryngopharyngeal sensory testing and was first described by Aviv et al. (1998, 2002). The quantitative testing of sensory thresholds is performed by endoscopically delivered standardized air puffs to the mucosa innervated by the superior laryngeal nerve to elicit the laryngeal adductor reflex. A study by Aviv et al. (2000) could show that the outcome of dysphagia management with regard to pneumonia incidence was the same using videofluoroscopy and FEESST.

6.2.5
Direct Endoscopy of the Pharynx and Larynx

Direct rigid endoscopy of the pharynx and larynx, which was developed by Kleinsasser (1968) ("laryngeal suspension microlaryngoscopy"), does not only allow microscopic evaluation of the pharynx and larynx, but also surgical therapies (phonosurgery with the aim of voice improvement, cold steel and laser surgery). The procedure is carried out under general anaesthesia. Various laryngoscopes are available in different sizes and types, e.g. from Kleinsasser (1968) (Fig. 6.6). The patient is lying in supine position. After protecting the teeth, the laryngoscope is inserted down to the level of the vocal folds under inspection of the hypopharynx and supraglottis. Then, the laryngoscope is held by a laryngoscope holder that rests on a table over or directly on the patient's chest. The microscope is then positioned.

Fig. 6.6. Laryngoscope according to Kleinsasser for direct microlaryngoscopy

Recently developed ventilation techniques have led to the possibility of tubeless jet ventilation to avoid intubation (Aloy et al. 1995). This method improves the operative conditions for the surgeon by providing more space for manipulation and better visibility. Moreover, it is also suited for laryngeal laser surgery, thus avoiding flammable tubes, and for endoscopic surgery of stenoses.

6.2.5.1
Examples of Typical Findings

Vocal Fold Granuloma. Figure 6.7 shows the microlaryngoscopical view of a typical vocal fold granuloma, which is located on the vocal process. It may occur after intubation (intubation granuloma) or is often associated with reflux disease (contact granuloma). Additional risk factors for development of a contact granuloma are functional voice disorders and psychogenic factors. For therapy, conservative treatment with proton pump inhibitors and logopaedic voice therapy can be tried. If the pathology persists or if a histological diagnosis is necessary, microlaryngoscopic surgery is performed.

Zenker's Diverticulum. As an alternative to the external approach with resection of a Zenker's diverticulum, endoscopic laser surgery may be performed. In Fig. 6.8, the party wall between the esophagus and Zenker's diverticulum is seen when endoscopically exposed before laser surgery.

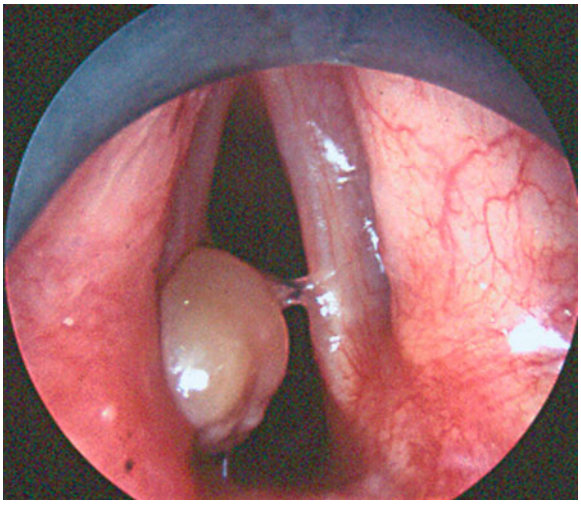


Fig. 6.7. Granuloma of the left vocal process (direct microlaryngoscopic view)

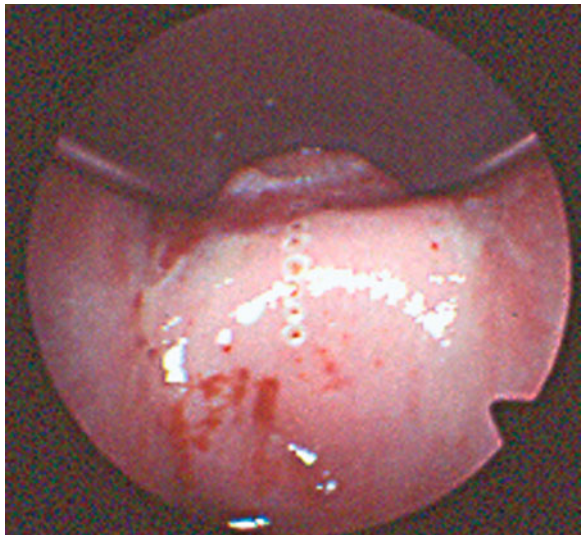


Fig. 6.8. Zenker's diverticulum (direct hypopharyngoscopic view), intraoperative view on a party wall between the esophagus and Zenker's diverticulum. The laser marking for the planned laser resection can be seen

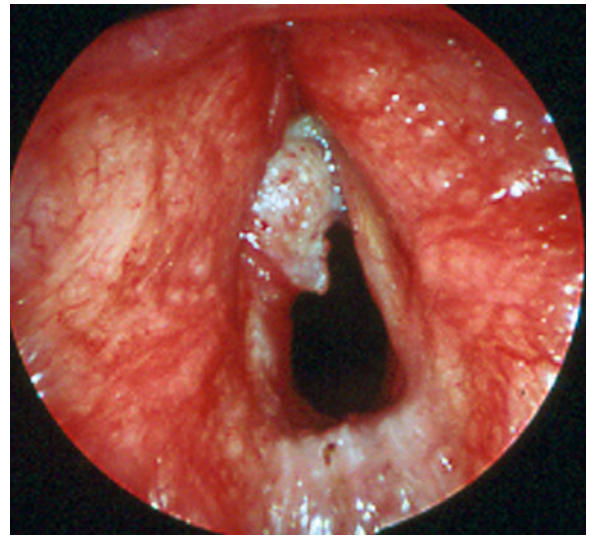


Fig. 6.9. Carcinoma of the larynx (direct microlaryngoscopy). The tumour mass is seen on the vocal folds

Laryngeal Carcinoma. An irregular mucosal surface or a tumour mass may be observed in the supraglottic (Fig. 6.9), glottic or subglottic area. Vocal fold motility may be impaired. Depending on the tumour size, the airway may be compromised.

6.2.5.2
Recent Developments and Future Aspects

New technologies try to enhance the endoscopic information during endoscopy.

Autofluorescence Endoscopy. The aim of this development is to enhance the endoscopic information during microlaryngoscopy. Autofluorescence is induced by filtered blue light of a xenon short arc lamp and processed by a CCD camera system. During microlaryngoscopy, the use of autofluorescence can improve the early detection and preoperative assessment of laryngeal cancer and its precursor lesions (Malzahn et al. 2002).

Videopharyngolaryngoscopy. The videoendoscopic technique has already become clinical standard for esophagogastroscopes. Now, technical progress has made video-rhinolaryngoscopes with a small diameter (about 3.9 mm) possible for transnasal insertion (Kawaida et al. 2002). The image transfer is not performed via the fibres, but with a chip camera at the tip of the endoscope. This technique provides better optical resolution and digital signal modulation. In the near future, the system will also allow videostroboscopy.

Contact Endoscopy. Contact endoscopy tries to improve the assessment of benign, premalignant and malignant pathologies of the larynx during microlaryngoscopy. The aim is to make epithelial cells visible, as in gynaecology. After staining the tissue with methylene blue, the magnifications obtained with contact endoscopy (60× and 150×) enable observation of the epithelium cells and their characteristics (Andrea et al. 1995).

3D Endoscopy. For scientific purposes, 3D endoscopic techniques were used in microlaryngeal surgery using tubeless jet ventilation (Schragl et al. 1995). This technique has not yet become clinical routine.

6.3 Endoscopy of the Esophagus

Endoscopy of the esophagus as part of the endoscopic evaluation of the upper gastrointestinal tract is one of the most frequent procedures performed in Western health care systems (Owings and Kozak 1998). The aim of esophagoscopy is diagnosis, differential diagnosis and follow-up of esophageal diseases. Moreover,

endoscopy supports further diagnostic procedures, such as endosonography, and enables therapeutic interventions. Two forms of esophagoscopy are in use: rigid and flexible endoscopy. Gastroenterologists and surgeons are used to flexible gastroscopes to perform total esophagogastroduodenoscopy, whereas otorhinolaryngologists prefer rigid instruments.

6.3.1 Symptoms of Esophageal Diseases

As the esophagus provides the bolus transport in the esophageal phase of swallowing, diseases of the esophagus bring about symptoms related to swallowing function. No single symptom is typical of a specific disorder. The localization of symptoms by the patient is unreliable. Patients who suffer from esophageal diseases (Table 6.1) may report the following symptoms (see also Chap. 2):

- *Dysphagia*: in the case of esophageal stenosis (e.g. esophageal carcinoma) or functional motility disorders (e.g. achalasia of the lower esophageal sphincter), the bolus transport is disturbed and causes the feeling of a stop of the bolus passage especially for solid food. The symptom dysphagia needs the analysis of all the four phases of deglutition (oral preparatory, oral, pharyngeal and esophageal phase), since oropharyngeal and esophageal dysphagia may influence each other or occur in combination.
- *Regurgitation:* reflux of swallowed bolus material from the esophagus to the pharynx and mouth due to retrograde esophageal motility, stenosing esophageal diseases or retained material (e.g. in Zenker's diverticulum).
- *Odynophagia:* painful swallow.
- *Globus sensation* (globus pharyngeus): globus sensation often derives from gastro-esophageal reflux disease. Other possible underlying causes that have to be considered for differential diagnosis are diverticula, webs, rings, but also thyroid gland diseases, cervical spine syndrome or functional voice disorders.
- *Heartburn*, *retrocardiac chest pain*: these symptoms occur in gastro-esophageal reflux disease, but also in esophageal carcinoma, esophageal spasms or esophagitis of other aetiologies. In approximately 40% of patients suffering from reflux disease, the typical symptom of heartburn is lacking. Exclusion of ischemic heart disease, pericarditis, aortic dilatation and pleuritis is mandatory.

Table 6.1. Esophageal diseases (modified from Seiden in Parparella et al. 1991)

MOTILITY DISORDERS
Primary disorders
Achalasia
Diffuse esophageal spasm
Nutcracker esophagus
Non-specific dysfunction (hypertensive lower esophageal sphincter, diminished amplitude of esophageal peristalsis)
Secondary disorders
Scleroderma and other connective tissue disorders
Diabetes mellitus
Alcoholism
Central nervous system disorders
Presbyesophagus
Chagas´disease
STRUCTURAL DISORDERS
Extrinsic compression
Webs, rings
Diverticula
Stricture due to reflux esophagitis
Ingestion of caustic substances
Hiatal hernia
Varices
Foreign bodies
Benign tumours
Malign tumours
Congenital disorders
Atresias
Tracheoesophageal fistulas
Duplications
Dysphagia lusoria
Achalasia

- *Cough* of unknown aetiology: cough may be due to aspiration or occur in esophageal reflux disease.
- *Gastrointestinal bleeding*: bleeding from the mouth without source in the nose, mouth, pharynx or larynx or overt/occult blood in the stool necessitates urgent esophagogastroduodenoscopy.

If one of these symptoms is present, endoscopy of the esophagus is indicated.

Esophagoscopy is further appropriate in (suspected) foreign body ingestion or as part of gastroduodenoscopy.

6.3.2 Rigid Esophagoscopy (Rigid Hypopharyngoesophagoscopy)

Traditionally, rigid (open tube) esophagoscopy is the method of choice for otorhinolaryngologists to remove foreign bodies located in the pharyngoesophageal segment or cervical esophagus and to perform tumour

staging (panendoscopy) in patients with primary malignancies in the head and neck to exclude/diagnose simultaneous additional malignancies (Dhooge et al. 1996). The incidence of simultaneous esophageal malignancies is about 8.4% (Dammer et al. 1999).

Usually rigid esophagoscopy is performed under general anaesthesia after informed consent. The patient is lying on his back, with the neck flexed and the head extended. The open tube esophagoscope (e.g. esophagoscope after Hasslinger) is inserted after protection of the teeth. Behind the arytenoids, the esophageal entrance is passed. The esophagoscope has to be advanced gently to avoid perforation. It is not possible to visualize the gastric mucosa safely with an open esophagoscope in all cases. If evaluation of the distal esophagus is needed, an esophagoscope with air insufflation can be used.

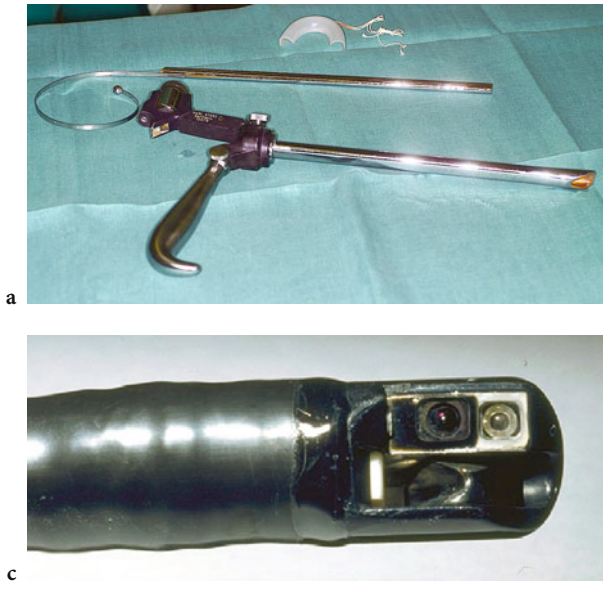
Risks include tooth damage, luxation of the arytenoids, bleeding and perforation of the hypopharynx or esophagus with consecutive mediastinitis or peritonitis. The complication rate lies under 1% (Schmidt et al. 1998).

6.3.3 Flexible Esophagoscopy

Esophagoscopy is performed for diagnosis of esophageal diseases, follow-up purposes, additional diagnostic procedures and for therapeutic measurements (hemostasis, dilatation, stenting, argon plasma coagulation, endoscopic mucosal resection).

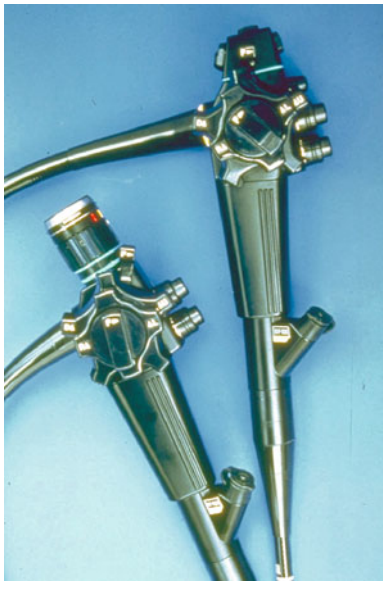
There are two types of endoscopes: the flexible fibre endoscope and the video endoscope. A channel in these endoscopes allows other instruments (forceps, brush, snare injection needle, dilatation balloon) to be passed through in order to take tissue samples, remove polyps, inject varices, dilate strictures or others. The length of the esophagogastroscope (Fig. 6.10) is about 100–120 cm, with a diameter of about 6–14 mm, depending on its purpose (ultra-thin stricture endoscope, therapeutic instruments with extra-thick channels). It has become standard to record the examination on a video recorder or file images in an electronic processing system for documentation. In the last two decades, the development of flexible video endoscopes with a CCD camera (charge coupled device), positioned on the tip, have revolutionized the image quality; already near to 1 million pixels are reached with chip technology.

Flexible endoscopy of the esophagus is usually performed on an outpatient basis in local anaesthesia using a spray containing benzocaine or tetracaine hydrochloride. The patient, who is kept without oral intake for 6–8 h prior to endoscopy, is offered intravenous sedation (e.g. with midazolam or propofol). He is placed in the left lateral decubitus position. After a hollow mouth piece is introduced, the lubricated endoscope is inserted under visual control. In the case of pathological or unclear findings in hypopharynx or larynx, the patient has to be referred to the otorhinolaryngologist. The instrument is advanced until the tip of the endoscope reaches the gastroesophageal junction (approximately 40 cm from the incisors). For examination of the stomach and



a

c



b

Fig. 6.10a–c. a Rigid esophagoscope. **b, c** Flexible gastroscope; control part (**b**), tip (**c**)

duodenum (flexible esophagoduodenoscopy), the tip is further advanced through the cardia, and the different portions of the stomach (cardia, fundus, corpus with greater and lesser curvature, antrum) are inspected. Afterwards, the tip is passed through the pylorus, into the duodenal bulb and the descending part of the duodenum.

The examination evaluates the lumen, wall, contents, peristalsis, appearance of the mucosal surface and visualizes/excludes flat, protruded or excavated lesions. If indicated, biopsies and brushing for histological, cytological and bacteriological examinations are performed.

Large clinical series report an incidence of moderate or severe complications in 0.1%–0.2%, mortality lies between 1 in 100,000 and 1 in 5,000, depending on the severity and urgency of underlying diseases and the proportion of therapeutic procedures. The following complications may occur: perforation, bleeding, cardiopulmonary complications, aspiration, side-effects of premedication, infection.

6.3.3.1 Examples of Typical Findings

Normal esophagus. The esophageal mucosa (non-keratinizing stratified squamous epithelium) appears pale, whereas the gastric mucosa is reddish (columnar epithelium). The transition between these two types of epithelium (esophagogastric junction) should be well visible. Because of its saw-toothed pattern, it is called z-line (Fig. 6.11).

Reflux esophagitis. Among the numerous patients with reflux symptoms, endoscopy can define the subgroup of those with reflux esophagitis characterized by reddening, erosions, ulceration or stricture at and above the z-line. A grading of reflux esophagitis can be given with the Savary and Miller (1977), the MUSE (metaplasia, ulcer, stricture, erosions) or the Los Angeles Classification. According to the grading by Savary and Miller (1977), four or five subgroups are described:

Grade 1, singular erosions; grade 2, confluent erosions; grade 3, esophagus is covered by circular erosions; grade 4, complications with peptic stricture, with or without signs of inflammation, or ulceration; grade 5, Barrett's esophagus. Figure 6.12 shows a minimal reflux esophagitis, Fig. 6.13 a severe reflux esophagitis with stricture.

Barrett's Esophagus. Due to long-lasting peptic reflux, the squamocolumnar junction in the distal esophagus moves upwards and the squamous epithelium is replaced by a specialized columnar epithelium with intestinal metaplasia. Barrett's metaplasia (Fig. 6.14) is a well-known risk factor for the development of dysplasia and adenocarcinoma (Fig. 6.15, early adenocarcinoma in Barrett's esophagus). Endoscopically, metaplastic gastric mucosa is recognized in the esophagus because of its salmon red appearance. Therefore, follow-up examinations with biopsies are necessary. For improving the diagnostic yield, chromoendoscopy, zoom endoscopy or fluorescence endoscopy can be added to the routine procedure.

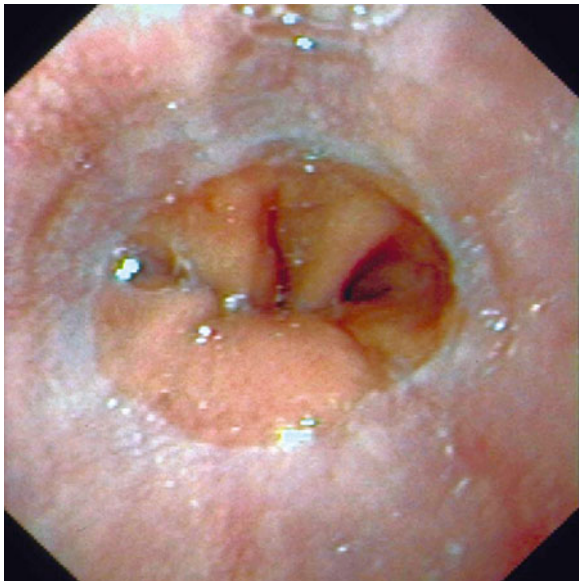


Fig. 6.11. Esophagogastric junction without pathology

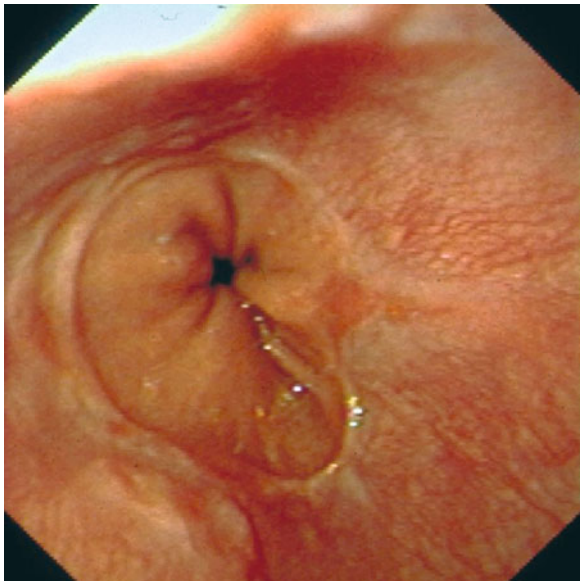


Fig. 6.12. Minimal reflux esophagitis

Esophageal carcinoma. Endoscopy shows early cancer (Fig. 6.16) or a polypoid or ulcerated mass or infiltration that can obstruct the esophageal lumen (Fig. 6.17). Multiple biopsies are taken for histological diagnosis. The incidence of adenocarcinomas derived from Barrett's esophagus increases in the USA and Europe dramatically, whereas the alcohol and tobacco associated squamous cell carcinoma becomes less frequent. In the case of a symptomatic tumour stricture, a balloon dilatation can be performed (Fig. 6.18) and a metal stent positioned as a palliative measure (Fig. 6.19).

Schatzki Ring. Endoscopy reveals a stricturing membrane in the distal esophagus. It may cause dysphagia, especially concerning solid food, and give rise to an impacted foreign body. For therapy, dilatation or thermal ablation during endoscopy is performed.

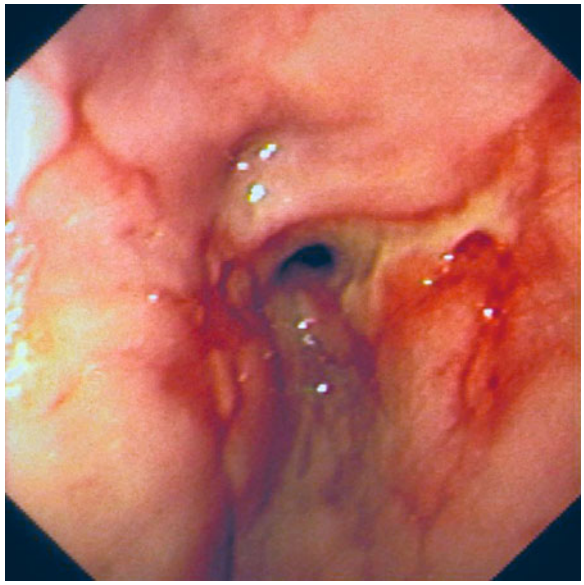


Fig. 6.13. Severe reflux esophagitis with stricture [grade 4 according to Savary and Miller (1977)]

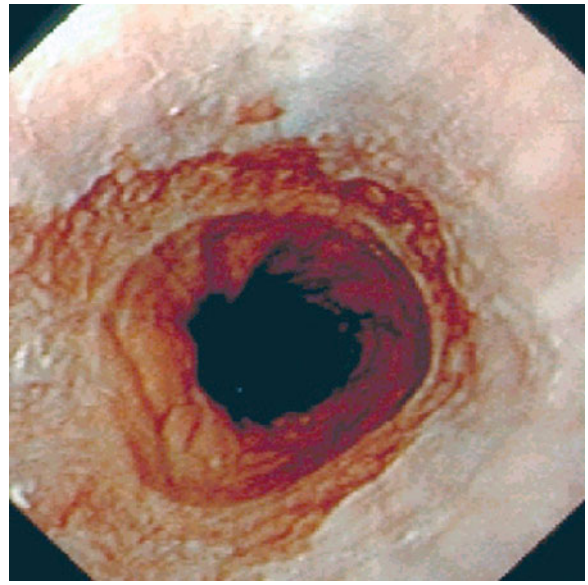


Fig. 6.14. Metaplasia due to chronic reflux (Barrett's esophagus)

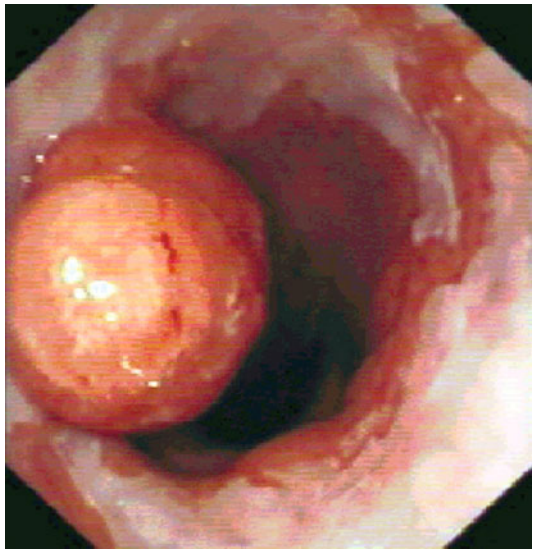


Fig. 6.15. Early adenocarcinoma in Barrett's esophagus

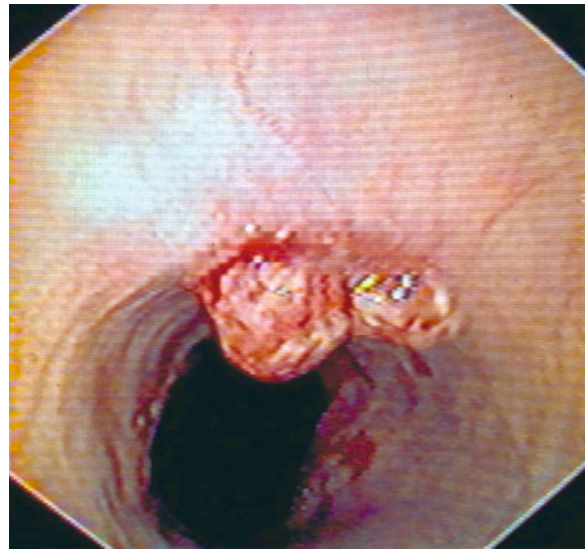


Fig. 6.16. Early squamous cell carcinoma of the mid-esophagus

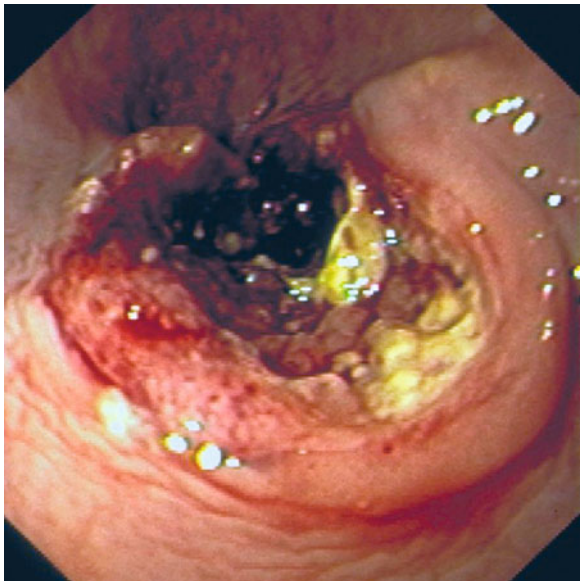


Fig. 6.17. Advanced ulcerated squamous cell carcinoma of the distal esophagus

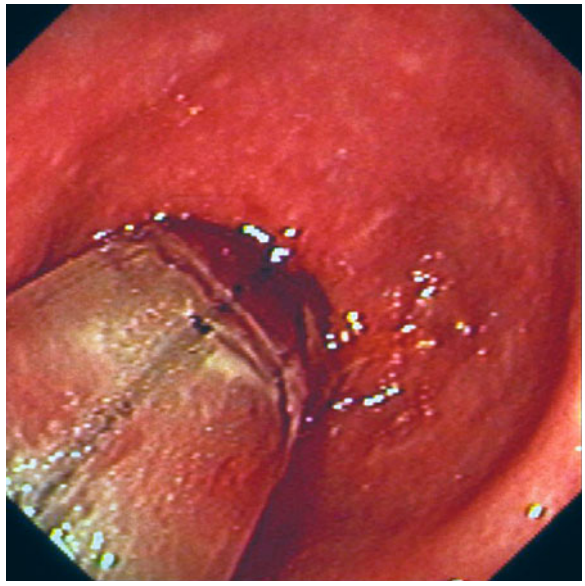


Fig. 6.18. Balloon dilatation of an esophageal stricture

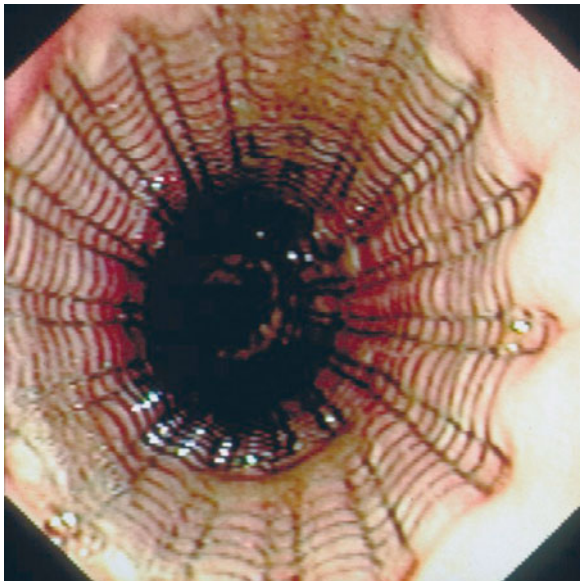


Fig. 6.19. Metal stent as palliative treatment of a malignant stricture

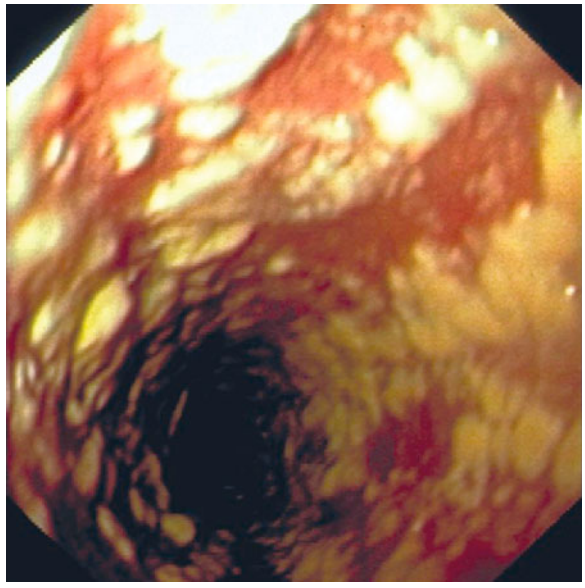


Fig. 6.20. Soor esophagitis

Soor esophagitis. A white coverage or single white spots (Fig. 6.20) are seen on the esophageal wall that can be removed with a forceps, but not with rinsing. Brush cytology easily depicts *Candida* during microscopic examination.

Esophageal web. Esophageal webs may be due to reflux disease, iron deficiency anaemia (Plummer Vinson syndrome) or idiopathic. They are either destroyed when passing the endoscope or removed with dilatation or bougienage.

Achalasia. Achalasia is a neuromuscular disorder of the esophagus that is characterized by a delayed esophageal emptying due to inadequate esophageal peristalsis and a non-relaxing, hypertensive lower esophageal sphincter. Diagnostic method of choice is videofluoroscopy and manometry, but endoscopy is necessary to rule out

other causes of dysphagia. It shows a dilated esophagus, weak non-propulsive esophageal peristalsis and retention of secretion and food. Endoscopic ultrasound reveals a thickened hypertrophic muscle layer of the esophagogastric sphincter. Endoscopy can also be used for therapeutic purposes, as pneumatic dilatation or botulinum toxin injection.

6.3.3.2 Endoscopic Ultrasound

The combined endoscopic and sonographic evaluation of the esophagus (endoscopic ultrasound, EUS) allows the identification of the different layers of the esophageal wall and contributes to the staging of esophageal tumours and locoregional nodal involvement (Tio 1998; Bergman and Fockens 1999; Richards et al. 2000; Wakelin et al. 2002). EUS proved to have a better accuracy in staging, especially in T1 and T2 tumours, than computed tomography (Ziegler et al. 1988; Tio 1998). Recently, high frequency EUS has been performed in early esophageal cancer to select patients for local endoscopic treatment.

EUS is a valuable tool to differentiate and further define submucosal tumours and extrinsic compression.

6.3.4. Rigid Versus Flexible Esophagoscopy

Both methods are less competing than substituting each other (Hörmann and Schmidt 1998): flexible equipment provides better imaging and allows air insufflation for distension, whereas rigid endoscopes facilitate instrumentation. Traditionally, otorhinolaryngology focuses on rigid hypopharyngo-esophagoscopy for removing foreign bodies and for panendoscopy in patients with malignant tumours in the upper aerodigestive tract to reveal additional simultaneous malignancies. Gastroenterology uses the flexible endoscope not only for the endoscopic evaluation of the esophagus, but also of the stomach and duodenum (esophagogastroduodenoscopy, EGD).

Rigid esophagoscopy is superior to flexible endoscopy in the evaluation of the hypopharynx and cervical esophagus. The skill and experience of the examiner remains of utmost importance (Monnier and Lang 1997). Flexible esophagoscopy does not allow a distinct examination of the upper esophageal sphincter region. In case of suspected malignancy in this region, rigid endoscopy should be performed. As foreign bodies mostly occur in the proximal esophagus, rigid endoscopy is an adequate procedure for the management of (suspected) foreign body ingestion (Alberty et al. 2001). Due to a higher perforation risk in the distal portion of the esophagus with rigid esophagoscopy, foreign bodies in that part are often removed by flexible endoscopy.

6.3.5. Recent Developments and Future Aspects

The development of ultra-thin esophagogastroscopes with an outer diameter of 5–6 mm allows the transnasal insertion of the endoscope, which may give rise to a greater acceptability and less discomfort of the patient (transnasal esophagogastroscopy). *Chromoendoscopy* has become a valuable adjunct to flexible endoscopy in oncological indications. It remains uncertain if new technologies, like *zoom endoscopy*, *optical coherence tomography* or *confocal laser endoscopy*, will become established. For sure, molecular pathological analysis of specimens will be of great clinical importance in the future.

6.4 Role of Endoscopy in the Diagnostic Work-Up of Dysphagic Patients

Differential diagnosis of dysphagia is based upon endoscopy and histopathological findings of biopsies, radiography, manometry and pH-metry. Radiology and endoscopy are both standard procedures which complement each other. Esophagoscopy is routinely performed to search for malignancy or to extract a foreign body. It is a method of first choice and capable of performing the differential diagnosis between structural or functional disorders. For motility disorders, videofluoroscopy, endoscopic ultrasound, manometry or pH-metry should be performed.

The advantage of radiographic studies is the identification of esophagotracheal fistulas, diverticula, atresia and hiatal/paraesophageal hernia. For radiological evaluation of dysphagia, the dynamic method of videofluoroscopy is regarded as the gold standard. However, subtle morphological changes are not visible radiographically.

The diagnostic indications for esophagoscopy are a matter of discussion: should endoscopy be performed primarily or not? Because of direct visualization, endoscopy is best for assessing mucosal integrity, inflammation and malignancies. Furthermore, it

enables biopsies to be taken for histological examination. Therefore, endoscopic follow-up is indicated in many diseases, e.g. Barrett's epithelium or achalasia.

In recent years, gastrointestinal endoscopy has gradually supplanted gastrointestinal radiography as the initial diagnostic study for the majority of patients with suspected gastrointestinal pathology. Technical developments (advances in lighting, imaging and flexibility) have improved the sensitivity and specificity and have made it a widely spread examination technique.

6.5 Conclusion

Endoscopy of the hypopharynx and esophagus contributes to the diagnostic work-up of the dysphagic patient. In many cases, it is the method of choice. However, radiography, especially videofluoroscopy, remains indispensable. For the future, the technical progress will stimulate and enable new endoscopic and radiographic developments. It aims at the highest possible quality of diagnosis and optimal patient acceptability.

References

Alberty J, Müller C, Stoll W (2001) Is the rigid hypopharyngoesophagoscopy for suspected body impaction still up to date. Laryngo Rhino Otol 80:682–686

Aloy A, Schachner M, Cancura W (1991) Tubeless translaryngeal superimposed jet ventilation. Eur Arch Otorhinolaryngol 248(8):475–478

Andrea M, Dias O, Santos A (1995) Contact endoscopy during microlaryngeal surgery. A new technique for endoscopic examination of the larynx. Ann Otol Rhinol Laryngol 104: 333–339

Aviv JE (2000) Prospective, randomized outcome study of endoscopy versus modified barium swallow in patients with dysphagia. Laryngoscope 110(4):563–574

Aviv JE, Kim T, Sacco RL, Kaplan S, Goodhart K, Diamond B, Close LG (1998) FEESST: a new bedside endoscopic test of the motor and sensory components of swallowing. Ann Otol Rhinol Laryngol 107:378–387

Aviv JE, Spitzer J, Cohen M, Ma G, Belafsky P, Close LG (2002) Laryngeal adductor reflex and pharyngeal squeeze as predictors of laryngeal penetration and aspiration. Laryngoscope 112(2):338–341

Bastian RW (1991) Videoendoscopic evaluation of patients with dysphagia: an adjunct to the modified barium swallow. Otolaryngol Head Neck Surg 104(3):339–350

Becker W, Naumann HH, Pfaltz CR (1983) Atlas der Hals-Nasen-Ohrenkrankheiten, 2nd edn. Thieme, Stuttgart

Bergman JJ, Fockens P (1999) Endoscopic ultrasonography in patients with gastro-esophageal cancer. Eur J Ultrasound 10(2–3):127–138

Bigenzahn W, Denk D-M (1999) Oropharyngeale Dysphagien. Ätiologie, Klinik, Diagnostik und Therapie von Schluckstörungen. Thieme, Stuttgart-New York

Dammer R, Bonkowski V, Kutz R, Friesenecker J, Schüsselbauer T (1999) Early diagnosis of additional tumors at diagnosis of primary oral carcinoma using panendoscopy. Mund Kiefer Gesichtschir 3:61–66

Denk D-M, Kaider A (1997) Videoendoscopic biofeedback: a simple method to improve the efficacy of swallowing rehabilitation of patients after head and neck surgery. Otorhinolaryngol 59:100–105

Dhooge IJ, De Vos M, Albers FW, Van Cauwenberge PB (1996) Panendoscopy as a screening procedure for simultaneous primary tumors in head and neck cancer. Eur Arch Otorhinolaryngol 253(6):319–324

Ekberg O, Olsson R (1997) Dynamic radiology of swallowing disorders. Endoscopy 29(6):439–446

Herrmann I (1998) Advanced course in Videopanendoscopy. 32. Fortbildungsveranstaltung für HNO-Ärzte, Hannover

Hörmann K, Schmidt H (1998) Flexible endosopy in ENT practice. HNO 46:654–659

Jones B, Donner MW (1991) The tailored examination. In: Jones B, Donner MW (eds) Normal and abnormal swallowing: imaging in diagnosis and therapy. Berlin, Springer, pp 33–50

Kawaida M, Fukuda H, Kohno N (2002) Digital image processing of laryngeal lesions by electronic videoendoscopy. Laryngoscope 112:559–564

Kleinsasser O (1968) Mikrolaryngoskopie und endolaryngeale Mikrochirurgie.Technik und typische Befunde. Schattauer, Stuttgart

Langmore SE (2001) Endoscopic evaluation and treatment of swallowing disorders. Thieme, Stuttgart, New York

Langmore SE, Schatz K, Olsen N. (1988) Fiberoptic endoscopic examination of swallowing safety: a new procedure. Dysphagia 2(4):216–219

Logemann J (1993) Manual for the videofluorographic study of swallowing, 2nd edn. Pro-ed, Texas

Logemann JA (1998) Evaluation and treatment of swallowing disorders. Pro-ed, Austin, Texas

Malzahn K, Dreyer T, Glanz H, Arens Ch (2002) Autofluorescence endoscopy in the diagnosis of early laryngeal cancer and its precursor lesions. Laryngoscope 112: 488–493

Monnier P, Lang FJ. (1997) Current position of endoscopy in otorhinolaryngology. HNO 45(11):886–887

Owings MF, Kozak LJ (1998) Ambulatory and inpatient procedure in the United States. Vital and Health Statistics. US-DHSS 139:1–13

Parparella MM, Shumrick DA, Gluckmann JL, Meyerhoff WL (1991) Otolaryngology. (vol 3: Head and Neck) WB Saunders Company, Philadelphia, London, Toronto, Montreal, Sydney, Tokyo

Richards DG, Brown TH, Manson JM (2000) Endoscopic ultrasound in the staging of tumours of the esophagus and gastro-esophageal junction. Ann R Coll Surg Engl 82(5):311–317

Savary M, Miller G (1977) Der Ösophagus. Lehrbuch und endoskopischer Atlas. Gassmann, Solothurn

Schima W, Denk D-M (1998) Videofluoroscopic and videoendoscopic studies: complementary methods for assessment of dysphagia. Proceedings, EGDG, Vienna

Schmidt H, Hormann K, Stasche N, Steiner W (1998) Tracheobronchoscopy and esophagoscopy in otorhinolaryngology. An assessment of current status HNO 46(7): 643–650

Schragl E, Bigenzahn W, Donner A, Gradwohl I, Aloy A (1995) Laryngeal surgery with 3-D technique. Early results with the jet-laryngoscope in superimposed high-frequency jet ventilation. Anaesthesist 44(1):48–53

Schröter-Morasch H (1999) Klinische Untersuchung des Oropharynx und videoendoskopische Untersuchung der Schluckfunktion. In: Bartolome G (Hrsg): Schluckstörungen. Diagnostik und Rehabilitation. 2. Aufl., Urban & Fischer, München-Jena, New York, pp 111–140

Schröter-Morasch H, Bartolome G, Troppmann N, Ziegler W (1999) Values and limitations of pharyngolaryngoscopy (transnasal, transoral) in patients with dysphagia.Folia Phoniatr Logop 51(4–5):172–182

Tio TL (1998) Endosonography in gastroenterology. Springer, Heidelberg

Wakelin SJ, Deans C, Crofts TJ, Allan PL, Plevris JN, Paterson-Brown S (2002) A comparison of computerised tomography, laparoscopic ultrasound and endoscopic ultrasound in the properative staging of osophago-gastric carcinoma. Eur J Radiol 41(2):161–167

Ziegler K, Sanft C. Semsch B, Friedrich M, Gregor M, Riecken EO (1988) Endosonography is superior to computed tomography in staging tumors of the esophagus and the cardia. Gastroenterology 94:A267

7 Cross-Sectional Imaging of the Hypopharynx and Esophagus

Christian Czerny, A. Ba-Ssalamah, Wolfgang Schima

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7.1 Introduction

In most cases, imaging of the hypopharynx and esophagus is performed in patients with swallowing disorders (Czerny and Formanek 2000; Schmalfuss 2002). The aetiology of swallowing disorders can be attributed to different entities, either congenital or acquired. In the pharynx, malignant tumours primarily cause swallowing disorders (Becker et al. 1998; Czerny and Formanek 2000). In the esophagus, swallowing disorders are often due to tumours, inflammatory strictures in gastro-esophageal reflux disease, or achalasia, and also may be due to either benign or malignant tumours. In most cases, the first choice for imaging evaluation is a double-contrast pharyngoesophagogram, which depicts nicely the morphology and extent of mucosal lesions. However, radiography (and endoscopy) are not suited to delineate the extramucosal tumour spread (into lymph nodes and adjacent organs) or abnormalities of the mediastinum in non-neoplastic diseases, such as Boerhaave syndrome or fistulas. In these patients, contrast-enhanced CT or MRI play an important diagnostic role in therapeutic management (Helmberger et al. 1996). Positron emission tomography (PET) may also be used if a malignant process is to be staged pre-therapeutically (Flamen et al. 2000; Junginger et al. 2002). The imaging techniques and pathologies of the hypopharynx and the esophagus differ, and therefore, these techniques and pathologies will be described separately.

C. Czerny, MD, Professor
A. Ba-Ssalamah, MD
W. Schima, MD, Professor
Department of Radiology, University of Vienna, Währinger Gürtel 18-20, 1090 Vienna, Austria

7.2 CT and MR Imaging Techniques of the Hypopharynx

7.2.1 CT

CT examination of the hypopharynx is performed in the axial plane in the helical CT mode (Kösling et al. 2000). All examinations should be performed using an intravenous injection of contrast material (Czerny and Formanek 2000). The amount of contrast material should be about 90–100 ml with a flow rate of 1.5–2 ml/s and a scan delay of 50 s. In a second scan to evaluate the piriform sinus, a Valsalva manoeuvre can be performed for better distension of the sinus.

The slice thickness, either on single-detector helical CT, should be about 3 mm and, on multidetector helical CT, the reconstructed slice thickness should also be about 3 mm. The window level setting should be a soft tissue window setting and a bone window setting.

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7.2.2 MRI

MRI of the hypopharynx is usually performed with a dedicated head-neck coil. Usually, an axial T2-weighted fast spin echo sequence with fat suppression or an inversion recovery (STIR) sequence can be used, followed by T1-weighted spin echo sequences in the axial plane before and after the IV application of contrast material (Schmalfuss 2002). Finally, a coronal T1-weighted sequence after the application of contrast material with fat suppression may be performed. The section thickness should not exceed 3–4 mm.

7.3 Pathologies of the Hypopharynx

7.3.1 Malignant Processes of the Hypopharynx

The predominant pathology of the hypopharynx is squamous cell carcinoma (Becker et al. 1998; Pameijer et al. 1998; Wenig et al. 1995). In most cases, these tumours are more or less asymptomatic in the earlier stages. Therefore, in most cases, these tumours are detected in the advanced stages (Table 7.1). Often there is an extension into the surrounding tissues, for instance, into the tongue base or the larynx or there is infiltration of the upper esophageal sphincter and the upper esophagus (Czerny and Formanek 2000). The squamous cell carcinomas show moderate to marked enhancement on contrast-enhanced CT images and also on T1-weighted spin echo images after contrast material application (Fig. 7.1). In most cases, these tumours show a mass effect.

In cases of piriform sinus tumours, a Valsalva manoeuvre may be helpful to obtain important diagnostic information for the therapeutic procedure because, particularly in this kind of tumour, the actual extension may not be quite clear from the "nonfunctional images", in many cases. Other malignant tumours, such as lymphomas, are somewhat rare and their cross-sectional imaging appearance is not quite characteristic. In the case of lymph node involvement, the lymph nodes of the lateral neck may be affected. The predominant group is lymph node section three, which is the middle jugular group.

Table 7.1. TNM staging of malignant tumours of the hypopharynx according to the American Joint Committee on Cancer Staging (AJCC)

Stage	Characteristic
TX	No primary tumour detectable
T0	No hint of primary tumour
Tis	Carcinoma in situ
T1	Tumour in one subregion
T2	Tumour infiltrating more than one subregion
T3	Tumour infiltrating more than one subregion with fixation of hemilarynx
T4	Tumour infiltrating surrounding structures

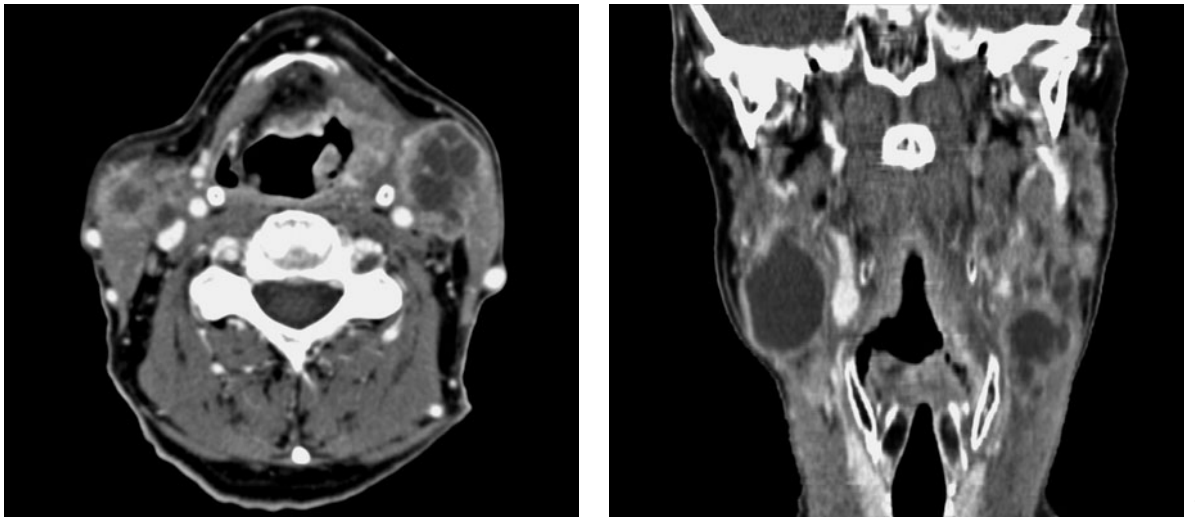
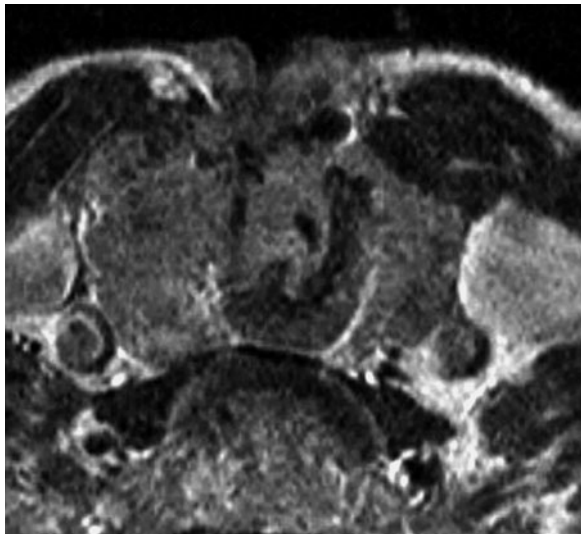


Fig. 7.1a,b. Hypopharyngeal carcinoma. **a** Axial contrast-enhanced CT image shows enhancing tumour formation in the left and right hypopharynx with extension over the borders of the hypopharynx. Also note lymph node metastases on both sides of the neck. **b** Coronal multiplanar reconstruction shows the large hypopharyngeal carcinoma and the massive lymph node metastasis on both sides of the neck

7.3.2 Benign Processes of the Hypopharynx

The hypopharynx may be affected by inflammatory processes, such as abscesses (Vogl et al. 2000). Hypopharyngeal abscesses differ from retropharyngeal abscesses in that these abscesses are more inferiorly and laterally located. Clinical symptoms include swallowing disorders, which are usually much more intense than those in retropharyngeal abscesses. In cross-sectional imaging, either on contrast enhanced CT or contrast-enhanced MRI, a cystic structure with a thick, irregular enhancing wall may be delineated and is indicative of a hypopharyngeal abscess.

A very common benign process of the hypopharynx is Zenker's diverticulum (Youssefzadeh et al. 2000). These diverticula occur superiorly to the cricopharyngeus muscle. The clinical signs are swallowing disorders and a feeling of a lump in the throat. The primary examination to detect Zenker's diverticulum is a conventional barium radiography or videofluoroscopy. On contrast-enhanced CT, the diverticula may appear as a mass with a more or less hypodense content, sometimes mixed with air bubbles. For differentiation from an abscess, repetition of the scan after administration of an oral contrast agent may be helpful.



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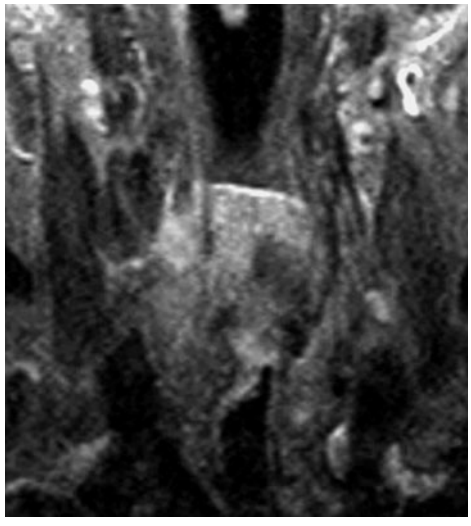


b

Fig. 7.2a,b. MRI of a recurrent hypopharyngeal carcinoma. **a** Axial contrast-enhanced T1-weighted spin echo image in high resolution shows tumour formation that enhances in the hypopharyngeal region. **b** Coronal contrast-enhanced T1-weighted spin-echo image in high resolution technique shows enhancing tumorous mass

7.3.3 Post-therapeutic Changes

In most cases, post-therapeutic cross-sectional imaging is performed to document the therapeutic process. The post-surgical cross-sectional imaging findings may vary from local laser or conventional tumour resection to complete laryngopharyngectomy and replacement by muscle or a small bowel reconstruction. In cases of radiotherapy, radiation-induced changes to the fatty tissue may be seen as mildly more dense linear structures within the fatty tissue. If the malignant tumour has completely disappeared after radiotherapy, stranding of the soft tissues may be the only persistent finding on CT scans. Residual tumour or recurrence may be delineated by a moderately to markedly enhancing mass with interval change during follow-up (Fig. 7.2).

7.4 CT and MR Imaging Techniques for the Esophagus

7.4.1 CT

CT of the esophagus is usually performed in conjunction with a CT of the chest, with the patient placed supine on the CT table (Okuda et al. 2002; Steinkamp et al. 1993). Before performing the CT examination, the esophagus should be delineated by oral contrast material and the application should be performed immediately before scanning. Typically, a dedicated low-density barium suspension for CT is used. However, if there is danger of aspiration,

a solution of an iodinated, water-soluble contrast agent should be used. With conventional helical CT, 80–100 ml of non-ionic contrast material with a flow of 2 ml/s and a scan delay of 30–40 s should be applied. Section thickness should be 3–5 mm, and a reconstructed section thickness for multi-detector helical CT, if used, should be about a 3-mm thickness. The advantage of multi-detector helical CT over single-detector helical CT is the capability of accurate 3D multiplanar reconstruction because of the availability of submillimeter detector configuration with isotropic voxels.

7.4.2 MRI

Usually MRI is performed with a dedicated body coil (Okuda et al. 2002; Steinkamp et al. 1993). After a localizer scan, a T2-weighted fast-spine echo sequence with fat suppression or an inversion recovery sequence with a fast spin echo technique may be performed in the coronal or axial plane, depending on the extension and localization of the process. Then, a T1-weighted spin echo sequence before and after intravenous application of gadolinium chelates is performed either in the axial or coronal plane, and a T1-weighted spin echo sequence after the application of intravenous contrast material with fat suppression either in the axial or coronal plane.

7.5 Abnormalities of the Esophagus

7.5.1 Carcinoma of the Esophagus

The most common malignant tumour of the esophagus is squamous cell carcinoma, followed by adenocarcinoma, while other types of carcinomas are rare (Lerut et al. 2001; Maier et al. 2000; Meyenberger and Fantin 2000; Tsuchiya et al. 1999). The predominant age for the appearance of squamous cell carcinoma is about the sixth decade and men are more commonly affected by this entity than women. Adenocarcinomas are most common in the distal esophagus, often presenting as a complication of long-standing gastroesophageal reflux disease. Yet, adenocarcinomas can occur in any part of the esophagus. In many cases, squamous cell carcinomas of the hypopharynx extend to the upper esophagus. An important complication to note is the infiltration of surrounding structures and the detection of lymph node metastasis, which are most commonly paraesophageal, mediastinal, and cervical. Mediastinal lymph nodes larger than 10 mm in short axis and with inhomogeneous or rim-like contrast enhancement may be highly suspicious for metastatic lymph nodes. Retrocrural lymph nodes should not exceed 6 mm in diameter. TNM staging of esophageal carcinoma is summarized in Table 7.2.

Carcinomas of the esophagus may be demonstrated as either a (sometimes bulgy) thickening of the wall, which typically extends a short distance, while inflammatory processes are, in some cases, less thickened and extend a greater distance (Fig. 7.3). The carcinoma may show an uptake of contrast material, which may be useful to differentiate the tumour from the surrounding tissue (Fig. 7.4). The surrounding periesophageal fatty tissue may delineate the tumour wall from other surrounding tissues. There are no established CT criteria to differentiate between tumours limited to the mucosa and tumours with extension into the muscularis propria. The infiltration of the surrounding tissues (T3) may be suspected if there is obliteration of the fat planes around the tumour. However, this CT sign is not sensitive, because fat planes may be lacking in certain anatomic regions, such as the interface between the esophagus and aorta. A T4 stage with invasion into adjacent organs is suspected if there is a compression of the airways or a mass within the lumen of the airways, or by the delineation of a fistula, which may be detected by small air bubbles in the mediastinum (Fig. 7.5). Esophageal cancer in the middle or lower third of the esophagus may invade into the descending aorta. The CT diagnosis of aortic invasion by cancer is based on the aortic circumference showing tumour contact. Tumour contact with ≥90° aortic circumference is highly suggestive of aortic invasion, whereas <45° vessel–tumour contact renders aortic invasion unlikely (Picus et al. 1983). CT diagnosis is indeterminate between 45° and 90°.

Table 7.2. Tumour staging of esophageal carcinoma

TNM Staging	
T1	Tumour <5 cm in longitudinal extension, no circular wall infiltration
T2	Tumour >5 cm in longitudinal extension, circular or obstructing tumour
T3	Infiltration of the surrounding fatty tissue
T4	Infiltration of surrounding structures
N1	Regional lymph nodes (cervical, mediastinal, perigastric)
M1	Distant metastases

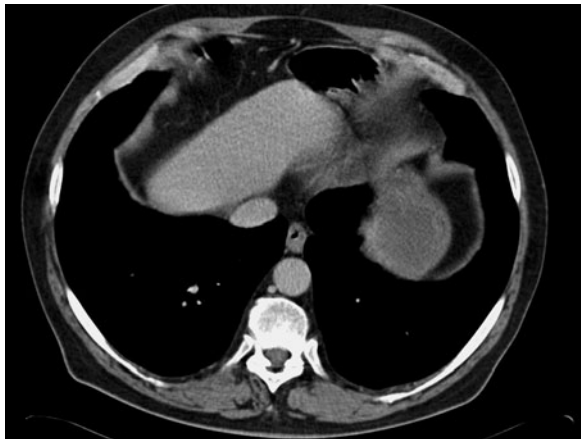


Fig. 7.3. Esophageal cancer, pT2 stage. Contrast-enhanced axial CT shows focal thickening of the distal third of the posterior esophageal wall. The fat planes surrounding the esophagus are intact. This finding is predictive of a T2 stage tumour, which was confirmed at surgery

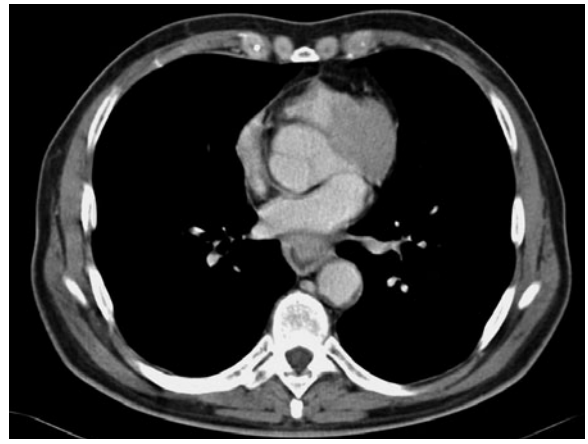


Fig. 7.4. Esophageal cancer, pT3 stage. Contrast-enhanced axial CT shows a markedly circumferential thickening of the esophageal wall with apparent obliteration of surrounding fat planes without infiltration of the adjacent structures, suggestive of a T3 stage tumour, which was confirmed at surgery

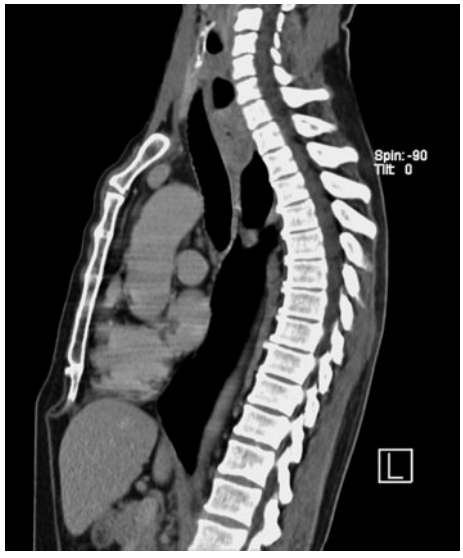


Fig. 7.5. Esophageal cancer, pT4 stage. Contrast-enhanced multidetector CT. Sagittal view shows a very large tumour of the third distal esophagus that extends beyond the esophageal wall and indents the gastroesophageal junction. This finding is indicative of gastric invasion. CT demonstrates lymph adenopathy in the perigastric region. This finding is predictive of a T4N1 stage tumour, which was confirmed at surgery

Other malignant tumours that may involve the esophagus are primary malignant melanomas, lymphomas, or malignant gastrointestinal stromal tumours (formerly known as leiomyosarcomas) (Ruppert-Kohlmayr et al. 1999). CT features of these tumours are not typical enough to differentiate them from carcinoma. Mediastinal or lung tumours and hematogenous metastases may affect the esophagus. Tumour involvement may be seen by displacement of the esophagus with mild contour irregularities at barium esophagrams. Contrast-enhanced CT reveals tumour origin and the full extent of tumour involvement of the mediastinum leading to esophageal invasion (Fig. 7.6).

7.5.2 Benign Tumours

Benign tumours of the esophagus are rare and appear in most cases as leiomyomas or fibrovascular tumours (Yang et al. 2001) (Fig. 7.7). Most benign esophageal tumours – provided they do not reach a certain size – are asymptomatic and thus, these tumours are mostly detected incidentally (Fig. 7.8). Occasionally, benign tumours may cause dysphagia or bleeding.

Leiomyomas (benign gastrointestinal stromal tumour) are by far the most common benign tumour in the esophagus. Radiographically they appear as a smooth submucosal mass. Accordingly, the CT features of leiomyoma are a homogenous submucosal/intramural mass with a smooth contour (Fig. 7.7). Occasionally, giant leiomyomas may appear as a large circumferential esophageal mass (Fig. 7.8). CT differentiation of submucosal leiomyoma from mucosal tumours is difficult, because mucosal integrity is much better assessed by radiography or endoscopy.

Fibrovascular polyps are rare, benign intraluminal masses, which may grow to enormous sizes in the esophagus. They contain predominantly fibrovascu-

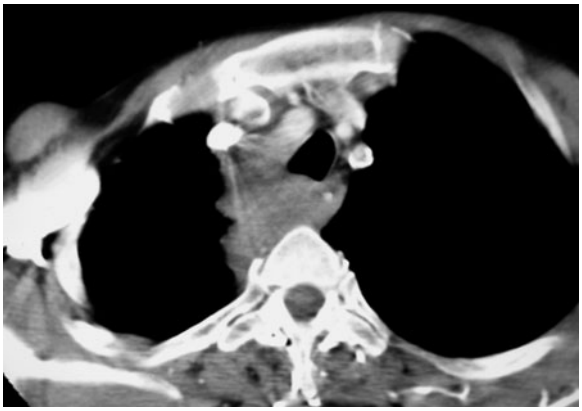


Fig. 7.6a,b. Lung cancer infiltrating the mediastinum and the esophagus. **a** Barium esophagram shows excessive deviation of the upper esophagus to the left due to a mediastinal mass. There is narrowing of the esophagus, but only mild contour irregularities typical of an extrinsic compression of the esophagus. **b** Chest CT demonstrates an apical lung cancer (Pancoast tumour) with infiltration into the mediastinum and esophagus

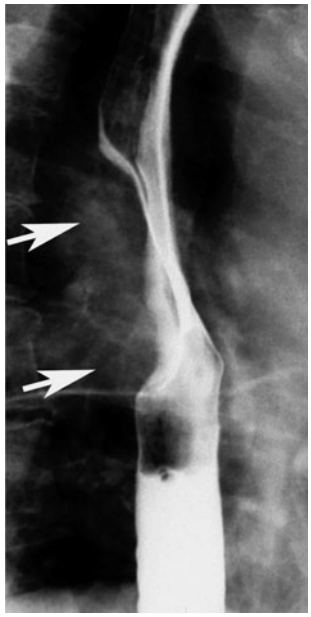


Fig. 7.7a,b. Leiomyoma. **a** Double contrast esophagram shows typical submucosal appearance of leiomyoma (*arrows*). **b** Contrast-enhanced CT after administration of oral contrast agent demonstrates a smooth submucosal mass. CT findings are suggestive of leiomyoma

lar and adipose tissue, which gives them their typical CT appearance of a pedunculated intraluminal mass of fat density, which expands the esophagus (Ascenti et al. 1999; Weiland et al. 2001). If fat tissue is predominant, MRI with T1-weighted pulse sequences with and without fat saturation may show the typical signal intensity decrease.

The esophagus is the least likely region of the gastrointestinal tract to be affected by hemangiomas or lipomas. At radiography, hemangiomas appear as

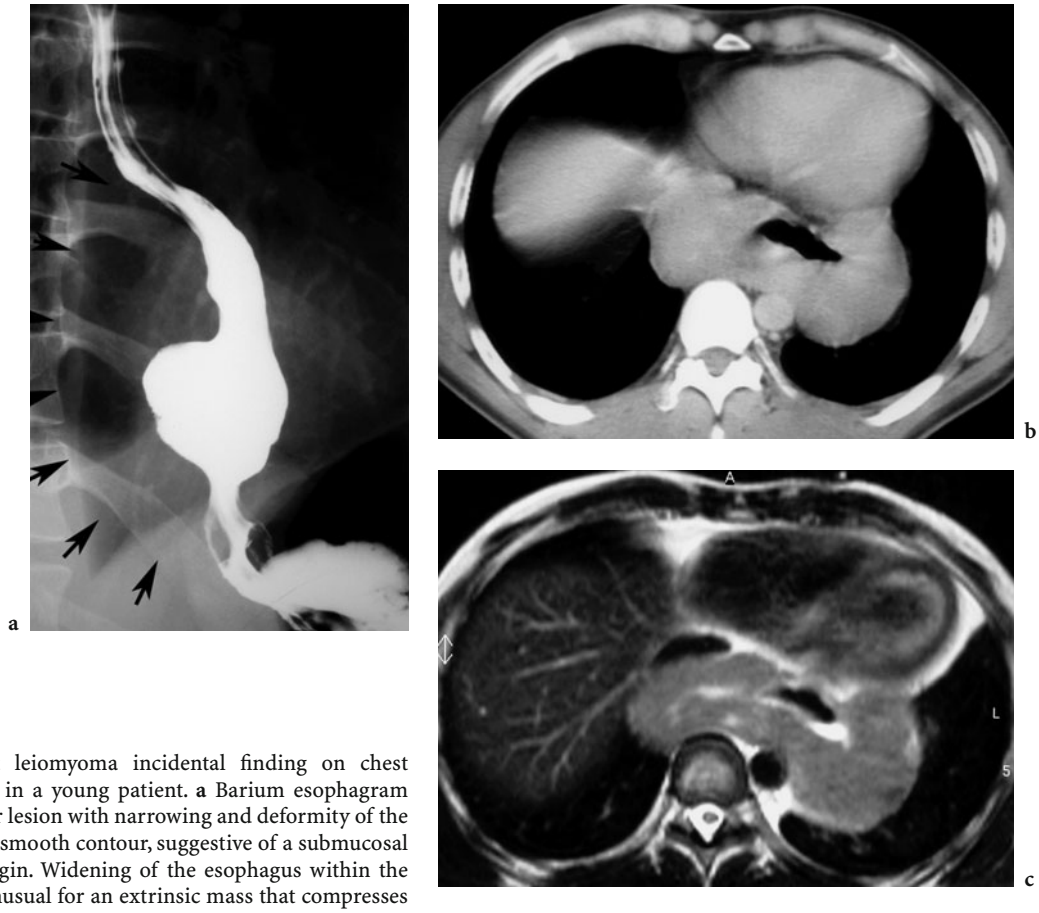


Fig. 7.8a–c. Giant leiomyoma incidental finding on chest film (not shown) in a young patient. **a** Barium esophagram reveals an annular lesion with narrowing and deformity of the esophagus with a smooth contour, suggestive of a submucosal or intramural origin. Widening of the esophagus within the mass would be unusual for an extrinsic mass that compresses the esophagus. *Arrows* indicate the size of the mediastinal mass as seen on the chest film. **b** CT reveals a large mass in the posterior mediastinum, growing circumferentially with widening of the esophagus. Findings are suggestive of an intramural mass. **c** Axial T2-weighted TSE MR image reveals a hypointense intramural mass of the esophagus

smooth or lobulated submucosal masses and are difficult to differentiate from other tumour. Cross-sectional imaging may be helpful: CT may demonstrate phleboliths in large hemangiomas and MRI features of very high signal intensity on T2-weighted images are suggestive of hemangioma.

However, the differentiation between benign tumours, such as leiomyomas, adenomas, or fibrovascular tumours (Ascenti et al. 1999; Weiland et al. 2001) from malignant tumours (e.g., lymphoma) is difficult with either CT or MRI and, in most cases, the appearance at cross-sectional imaging is not characteristic.

7.5.3 Fistulas of the Esophagus

Esophageal fistulas can be classified according to their anatomic relationship into esophageal-airway, esophago-pleural, aorto-esophageal and esophago-pericardial fistulas (Levine 2000). Esophageal-airway fistulas can be either congenital (so-called tracheo-esophageal fistulas) or acquired. Most esophageal-airway fistulas are a complication of tracheo-bronchial invasion by esophageal cancer. The likelihood of fistula formation is increased after radiation therapy of cancer. Other less common causes of fistula formation are trauma, foreign bodies or infection. CT is indicated to precisely localize the fistula and complications, such as abscesses in the mediastinum. Aorto-esophageal or esophago-pericardial fistulas are very rare, but have a very high mortality. Aorto-esophageal fistulas are either caused by intraesophageal rupture of an aortic aneurysm or by aortic injury of an esophageal foreign body (Lam et al. 2003). Patients may present with massive hematemesis and contrast-enhanced CT is the method of choice to show aortic rupture, although the fistula itself

may escape detection at CT. Esophago-pericardial fistulas are caused by cancer, foreign bodies, and ulceration of the esophagus in severe esophagitis (Cyrlak et al. 1983). Contrast studies and CT may show the combination of contrast extravasation and pneumopericardium, which is highly suggestive of esophago-pericardial fistula (Fig. 7.9).

7.5.4 Perforation of the Esophagus

Perforation of the esophagus is a life-threatening event that mandates a rapid diagnosis. In most cases, the cause of perforation includes foreign bodies, endoscopy or trauma. Tumours and strictures may also cause perforations.

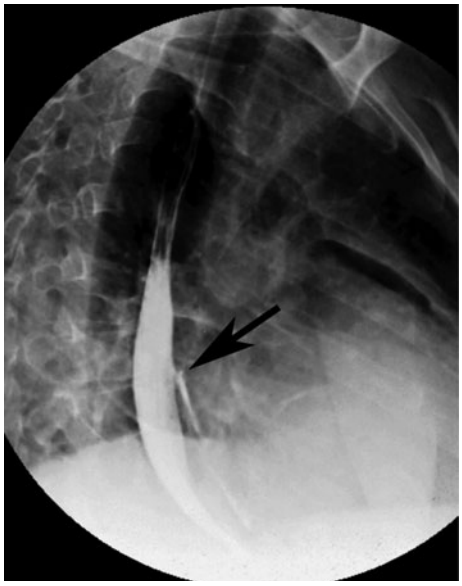
A special form of perforation of the esophagus is the so-called Boerhaave syndrome (Fig. 7.10). This rupture of the esophagus is caused by abnormal intraluminal pressure, caused by forceful vomiting. The rupture is usually located just cranial to the esophagogastric junction. Contrast studies are the method of choice to demonstrate esophageal rupture. However, contrast-enhanced CT should be performed in all patients in whom perforation is suspected. In patients with negative esophagrams, CT may demonstrate subtle signs of mediastinal emphysema, which is highly suspicious for perforations in this clinical situation. In patients with rupture shown by contrast studies, CT may show the amount of mediastinal or pleural fluid retentions (White et al. 1993).

7.5.5 Dysphagia Lusoria

A congenital anomaly, an aberrant right subclavian artery arising as the fourth arterial branch from the proximal descending aorta has been termed arteria lusoria. It courses posteriorly to the esophagus and causes a posterior indentation of the esophagus readily seen on barium esophagrams. In most individuals, an arteria lusoria remains asymptomatic, but dysphagia may be caused in a few (so-called dysphagia lusoria). On CT angiography an abnormal course of the vessels or an abnormality of the aortic arch may be well delineated (Fig. 7.11). Another imaging method to delineate these abnormalities is contrast-enhanced MR angiography.

7.5.6 Other Benign Entities

Other various benign entities of the esophagus may be *diverticula*, *varices*, *hiatal hernia*, and *strictures* or *stenoses*. Diagnosis of these lesions may be inciden-



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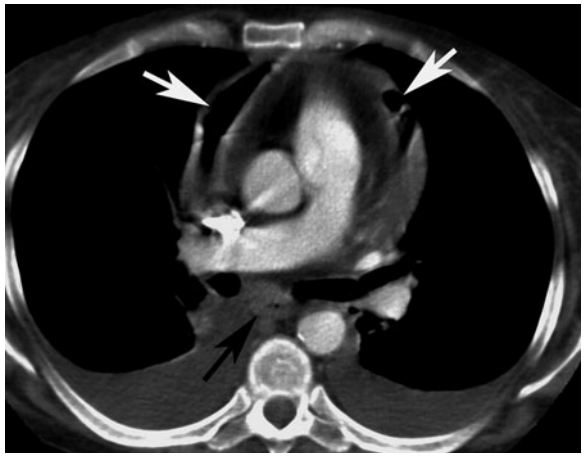

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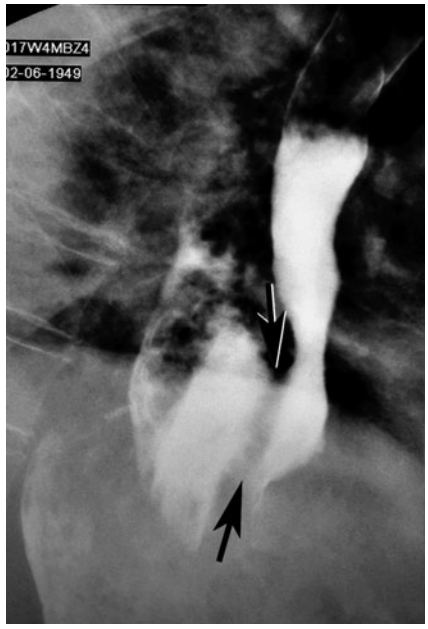
Fig. 7.9a,b. Esophago-pericardial fistula in a patient with esophageal ulceration. **a** Esophagram with water-soluble contrast shows a small fistula with linear extravasation of contrast (*arrow*). **b** Chest CT (obtained before esophagram) shows thickening of the esophagus (*black arrow*) and pneumopericardium (*white arrows*). However, the fistula is not depicted. Penetrating esophageal ulcer with esophago-pericardial fistula was confirmed at autopsy



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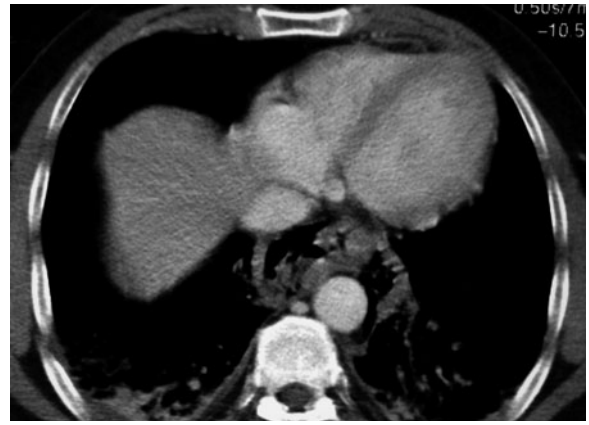

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Fig. 7.10a–c. Boerhaave syndrome in a patient with hyperemesis. **a** Esophagram with water-soluble contrast agent shows large rupture (*arrows*) of the lower esophagus with contrast extravasation into the mediastinum. **b** Contrast-enhanced CT (soft tissue window setting) demonstrates widening of the posterior mediastinum and air around the esophagus and the aorta. **c** CT with lung window settings reveals pneumomediastinum around the esophagus to a much greater extent

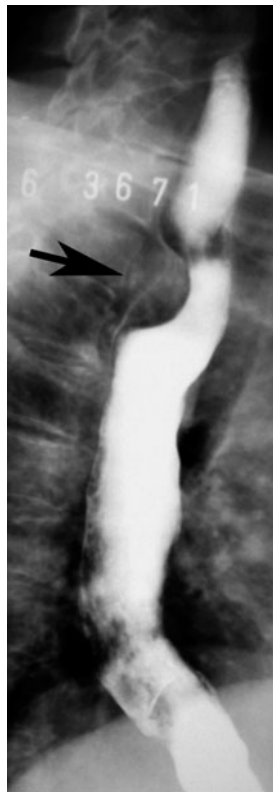
tally made in patients referred for chest/abdomen CT for other reasons. CT diagnosis of a benign entity should be reliably made to rule out the presence of a tumorous lesion. Especially in case of stenosis CT may be indicated to rule out the presence of tumour, when the results of endoscopic biopsy are equivocal and endosonography is not available.

On contrast-enhanced CT examination, a *diverticulum* of the esophagus is demonstrated as an outpouching that contains air bubbles and/or orally administered contrast material. The two predominant locations of esophageal diverticula are the mid-esophagus (at the level of the tracheal bifurcation) and the distal esophagus (so-called epiphrenic diverticula).

Varices of the esophagus are mainly caused by portal hypertension. In this case gastric varices communicate with the esophageal and periesophageal veins, which are drained via the azygos/hemiazygos venous system to the superior vena cava. Contrast-enhanced CT may be superior to barium radiography in the depiction of varices, as it may delineate periesophageal varices. On contrast-enhanced CT, varices appear as enhancing tortuous or tubular structures along the wall of the esophagus (Fig. 7.12). This can result in thickening of the wall and/or narrowing of the lumen of the esophagus.

Hiatal hernias are also primarily detected on thorax or abdominal CTs and are often detected incidentally (Polat et al. 2002). In most cases, hiatal hernias present as a mass structure superior to the diaphragm and retrocardially positioned, and contain air and soft tissue (Fig. 7.13). These hernias can by classified as either axial or paraesophageal hernias.

Inflammatory strictures or *circumscribed stenoses* may be a complication of esophagitis (e.g. reflux esophagitis), radiation therapy, trauma, surgery, or after lye ingestion. At CT, such strictures may present as a narrowing of the esophageal lumen, sometimes in conjunction with mild circumferential wall thickening of the esophagus. CT shows dilatation of the proximal esophagus with stasis of secretions or orally administered contrast material and distal tapering of the esophagus. CT can provide important information about the delineation or exclusion of an extraluminal mass as the underlying cause of stenosis.



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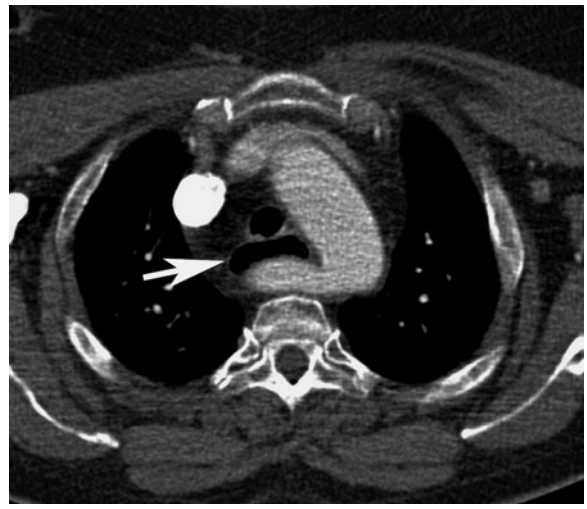

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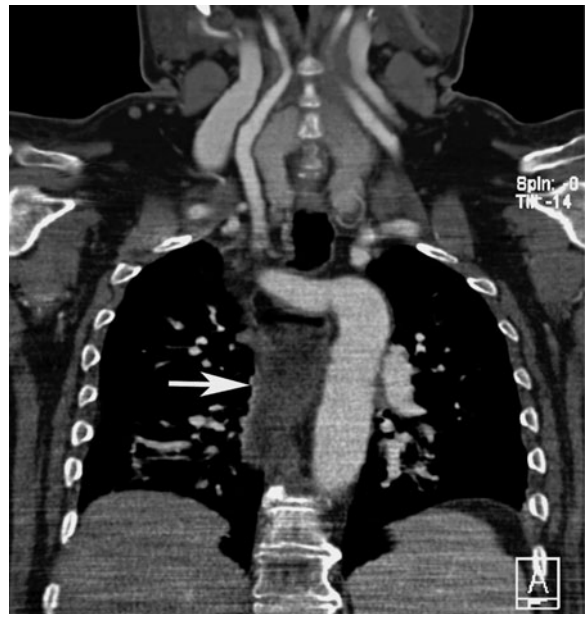

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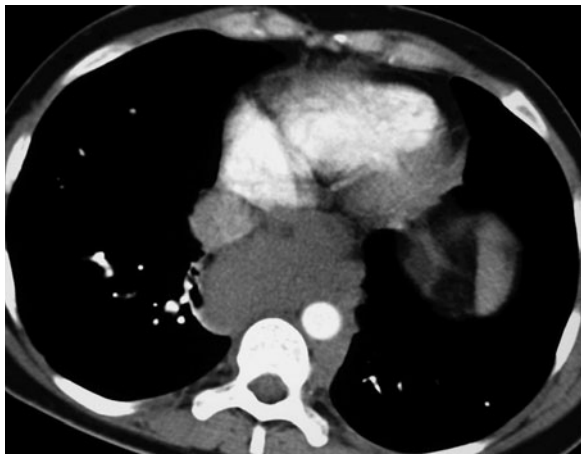
Fig. 7.11a–c. Arteria lusoria and achalasia in a patient suffering from dysphagia for 2 years. **a** Barium esophagram reveals a dilated esophagus in achalasia. However, esophageal dilatation is present cranially and caudally of posterior impression (*arrow*). **b** Multi-detector CT shows an aberrant right subclavian artery coursing posterior to the esophagus, typical for arteria lusoria. Esophagus is air-filled and dilated due to achalasia (*arrow*). **c** Coronal 3D reformation reveals arterial anatomy of arteria lusoria. There is dilatation of the esophagus, which is fluid-filled in the lower two-thirds (*arrow*). Late-onset dysphagia in this 60-year-old patient is most likely due to achalasia and not due to congenital arteria lusoria



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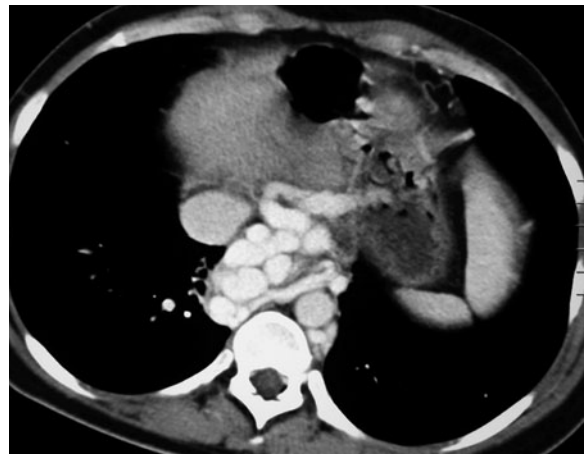

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Fig. 7.12a,b. Tumour-simulating esophageal varices in a patient with portal vein thrombosis. **a** Contrast-enhanced CT in the arterial-dominant phase shows a large low-density mass in the posterior mediastinum. **b** CT in the portal-venous phase shows intense contrast enhancement of large periesophageal varices

Fig. 7.13. Axial hiatal hernia. Contrast-enhanced multidetector CT. Coronal view shows marked fold thickening of the esophagogastric junction and an intrathoracic hernia. Note the smooth mucosal enhancement and clear fat planes around the esophagus

References

Ascenti G, Racchiusa S, Mazziotti S, Bottari M, Scribano E (1999) Giant fibrovascular polyp of the esophagus: CT and MR findings. Abdom Imaging 24:109–110

Becker M, Moulin G, Kurt AM, Zbaren P, Dulgerov P, Marchal F, Zanaret P, Lehmann W, Rufenacht DA, Terrier F (1998) Atypical squamous cell carcinoma of the larynx and hypopharynx: radiological features and pathologic correlation. Eur Radiol 8:1541–1551

Cyrlak D, Cohen AJ, Dana ER (1983) Esophagopericardial fistulas: causes and radiographic features. AJR 141:177–179

Czerny C, Formanek M (2000) Malignant tumors of the pharynx. Radiologe 40:625–631

Flamen P, Lerut A, Van-Cutsem E, De-Wever W, Peeters M, Stroobants S, Dupont P, Bormans G, Hiele M, De-Lexn P, Van-Raemdonck D, Coosemans W, Ectors N, Haustermans K, Mortelmans L (2000) Utility of positron emission tomography for the staging of patients with potentially operable esophageal carcinoma. J Clin Oncol 18:3202–3210

Helmberger R, Jager L, Grevers G, Reiser M (1996) Computerized tomography of malignancies of the oral cavity, the oropharynx, and hypopharynx and invasiveness. Radiologe 36:193–198

Junginger T, Kneist W, Schreckenberger M, Menzel C, Oberholzer K, Bartenstein P (2002) Positron emission tomography for the preoperative staging of esophageal carcinoma. Dtsch Med Wochenschr 127:1935–1941

Kösling S, Schmidtke M, Vothel F, Hahn S, Weidenbach H, Oeken J (2000) Value of spiral CT in the staging of carcinomas of the oral cavity, the oropharynx, and the hypopharynx. Radiologe 40:632–639

Lam EC, Brown JA, Whittaker JS (2003) Esophageal foreign body causing direct aortic injury. Can J Gastroenterol 17: 115–117

Lerut T, Coosemans W, De-Leyn P, Van-Raemdonck D, Nafteux P, Moons J (2001) Optimizing treatment of carcinoma of the esophagus and gastroesophageal junction. Surg Oncol Clin North Am 10:863–884

Levine MS (2000) Miscellaneous abnormalities of the esophagus. In: Gore RM, Levine MS (eds) Textbook of gastrointestinal radiology. W.B. Saunders, Philadelphia, pp 465–483

Maier A, Woltsche M, Fell B, Prettenhofer U, Domej W, Roger GM, Pinter H, Smolle-Juttner FM (2000) Local and systemic treatment in small cell carcinoma of the esophagus. Oncol Rep 7:187–192

Meyenberger C, Fantin AC (2000), Esophageal carcinoma: current staging strategies. Recent Results Cancer Res 155:63–72

Okuda I, Kokubo T, Hoshihara Y, Udagawa H (2002) Imaging diagnosis of esophageal carcinoma by computed tomography and magnetic resonance imaging. Nippon Geka Gakkai Zasshi 103:331–336

Pameijer FA, Mukheri SA, Balm AJ, Laan BF van der (1998) Imaging of squamous cell carcinoma of the hypophayrnx. Semin Ultrasound CT MR 19:476–491

Picus D, Balfe DM, Koehler RE et al. (1983) Computed tomography in the staging of esophageal carcinoma. Radiology 146:433-438

Polat P, Alper F, Kantarci M, Levent A (2002) Sliding hernia simulating esophageal tumor on computed tomography. Dis Esophagus 15:189–191

Ruppert-Kohlmayr AJ, Raith J, Friedrich G, Regauer S, Preidler KW, Szolar DH (1999) Giant liposarcoma of the esophagus: radiological findings. J Thor Imaging 14:316–319

Schmalfuss IM (2002) Imaging of the hypopharynx and cervical esophagus. Magn Reson Imaging Clin North Am 10:495–509

Steinkamp HJ, Heim T, Maurer J, Mathe F, Felix R (1993) The value of magnetic resonance tomography and computed tomography in the tumor staging of laryngeal and hypopharyngeal carcinomas. Röfo Fortschr Geb Roentgenstr Neuen Bildgeb Verfahr 58:73–444

Tsuchiya Y, Onda M, Miyashita M, Hagiwara N, Maruyama H, Matsutani T, Okawa K, Takubo K, Yamashita K, Sasajima K (1999) Combined modality therapy for basaloid squamous carcinoma of the esophagus. Hepatogastroenterology 46:2868–2871

Vogl TJ, Mack M, Balzer J, Diebold T (2000) Inflammatory diseases of the pharynx: Radiologic imaging and diagnostic methods. Radiologe 40:619–624

Weiland G, Knipping L, Mangold G (2001) Fibrovascular esophageal polyp-diagnosis and therapy. Chriurg 72: 847–852

Wenig BL, Ziffra KL, Mafee MF, Schild JA (1995) MR imaging of squamous cell carcinoma of the larynx and hypopharynx. Otolaryngol Clin North Am 28:609-619

White CS, Templeton PA, Attar S (1993) Esophageal perforation: CT findings. AJR 160:767–770

Yang PS, Lee KS, Lee SJ, Kim TS, Choo IW, Shim YM, Kim K, Kim Y (2001), Esophageal leiomyoma: radiologic findings in 12 patients. Korean J Radiol 2:132–137

Youssefzadeh S, Lomoschitz F, Czerny C, Cartellieri M (2000), Congenital and benign lesions related to the pharynx. Radiologe 40: 610–618

8 The Pediatric Esophagus

Jane E. Benson

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8.1 Introduction

Feeding one's child and watching him grow and thrive is one of the chief joys of parenting, so anything that interferes with this is apt to prompt a quick demand for medical attention. Symptoms such as vomiting, choking, irritability and food refusal are all very distressing to both parent and child. However, because the gastrointestinal (GI) tract in a child, particularly a young infant, is relatively short and compact, abnormalities at the level of the pharynx, esophagus, stomach, small bowel, or even colon can result in puzzlingly similar clinical manifestations. Determining a cause for the symptoms often falls to the radiologist, who has the unique opportunity to observe the GI tract in action.

J. E. Benson MD
Assistant Professor, Russell H. Morgan Department of Radiology and Radiological Science, The Johns Hopkins University School of Medicine, 5722 Oakshire Road, Baltimore, Maryland 21209, USA

8.2 Imaging Methodology and Techniques: Showing Anatomy and Function for Diagnostic Efficacy

The range of procedures available to image the pediatric esophagus is the same as that available for adults, but the choice is influenced by the odds of finding certain types of pathologies. Moreover, the application of these procedures differs from that in adults because of the unique challenge of obtaining a diagnostic examination in the pediatric patient.

8.2.1 Fluoroscopy

Fluoroscopy is the mainstay of esophageal examination, as it not only displays anatomy but, just as importantly, function. The area examined may be confined to the esophagus, but usually the esophagus is seen in the context of the entire upper GI tract, from mouth to ligament of Treitz. This examination is usually done with the child supine or in lateral position on the fluoroscopy table. Swallows in true lateral position are extremely important to look for contrast in the trachea and deformity or narrowing of the esophagus from the posterior or anterior aspect. True AP projections look for esophageal displacement or narrowing from the sides. Videotaping of the fluoroscopy is extremely helpful, as abnormalities may be fleeting.

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Volitional swallow and good distension of the esophagus are of paramount importance. These can only be achieved with a comfortable, cooperative patient. Infants and older children are easier to manage, while toddlers and young children are more apt to be frightened and less persuadable. Have the room as warm and as light as possible, and allow the child to keep his clothing on; remove only large metallic fastenings and add a protective gown. Put a pad or blanket on the fluoroscopy table. Allow the child to hold a favorite blanket or stuffed animal. Introduce the fluoroscopy image intensifier as a big "tent" or "house". Show the child how it will get very close to him but will not touch him. A video machine playing a children's movie or cartoon is an excellent distraction.

The choice of contrast can improve the chances of getting a good exam. Standard oral barium sulfate preparations have the consistency of milk and some sweetening with sorbitol or the like, and a very hungry infant is usually very cooperative and needs no additional persuasion. It is helpful to use the type of nipple that the infant is familiar with, and also to enlarge the endhole slightly. With an older toddler, hiding the contrast in a familiar cup with a straw can be successful. Barium can be flavored with different fruit drink powders or with chocolate syrup; give the child his choice. Water-soluble iodinated contrast has a bitter taste that is more difficult to disguise, and in the concentrations often necessary for it to be seen on fluoroscopy it is not as benign as barium if it is aspirated. In post-surgical examinations when there is risk of perforation of the esophagus, however, its use may be mandated.

There are times when the child refuses to cooperate. It is helpful at this point to "triage" the clinical questions: what is the most important question to be answered? The next most important? You must do this, because structuring the exam to answer one question may take away your chance to answer another. If the examination cannot be rescheduled for another day when the child might be more hungry or better rested, then the contrast must be administered manually. Assistance from parents and/or technologists is necessary. Wrapping the child's legs in a sheet can often help persuade him that resistance is futile. A small syringe (5cc for a toddler, 10cc for an older child) is used to put aliquots of contrast as far back in the child's mouth as possible. At least a small amount is generally swallowed, allowing appreciation of pharyngeal function and esophageal motility. If this fails, then a small feeding tube is passed through the child's nose and into the distal esophagus. With the child in lateral position, the radiologist pulls the tube back slowly, hand-injecting at intervals sufficient volume to distend the esophagus, until the entire esophagus is imaged. This gives a poor assessment of esophageal motility, however, and there is danger of aspiration. In addition, a "normal" study performed only with a tube will miss the pathology associated with swallowing that can be found in up to half the patients referred for examination (Vazquez and Buonomo 1999).

Assessing gastroesophageal reflux fluoroscopically takes patience. It is best to do this at the end of the examination. The child's stomach should be full and he should be relaxed and distracted. If the preceding part of the study has been unpleasant, the infant might be allowed to feed in the parent's arms until full, then replaced beneath the fluoroscope. Darkening the room and minimizing noise might encourage the child to fall asleep in a supine position. Videotapes provide solace and distraction for the toddler. Position the fluoroscope to show the esophagus, then image only for about 1 second out of every 7-10, for a total of 5 minutes (less than 1 minute of fluoro time). Significant reflux, i.e. reaching the clavicles, cervical esophagus or pharynx, will persist long enough to be recorded. Crying reinforces the diaphragmatic hiatus and minimizes reflux, so assessment in an inconsolable child may have to be abandoned.

The modified barium swallow is conducted very differently. Here, the child's usual feeding position is duplicated in a chair or seat, and graded food textures are offered, opacified with contrast. A trained feeding therapist should be in attendance who chooses the textures to be tested, and a familiar person should do the feeding. Because of the confines of the imaging chair and the fluoro image intensifier, imaging is usually done from the lateral projection only. The study is recorded on videotape alone. Because the child is less restrained, the radiologist must work to keep the pharynx centered in the screen.

8.2.2 Radiation Dose Reduction

Throughout any fluoroscopic exam, the radiologist must be constantly mindful of the accumulating radiation dose. Grids should not be used for children less than adult size. The thyroid has to be part of the field of view, but gonads, pelvic bone marrow, and the ocular lenses should be rigorously excluded by coning and shielding. Intermittent review of the videotape may find that pathology has been imaged that escaped

direct visualization, and that additional imaging is unnecessary. Pulsed fluoroscopy can dramatically reduce patient dose with relatively minor image degradation (Hernandez and Goodsitt 1996). Digital fluoroscopy units have the added advantage of "last image hold," whereby the image present on the screen when the examiner stops fluoroscoping remains there and can be captured as a permanent digital image. Since the examiner can react more quickly to seeing a pathological event than the spot film device can record it, there is a better chance that it will be caught. This also avoids the dose from spot-film technique.

8.2.3 Advanced Imaging Techniques

Nuclear medicine offers a low-dose way to monitor an entire feeding and post-prandial period to look for aspiration and reflux. Technetium 99m is the isotope of choice to label a meal. The limitation is not on imaging time, but rather on patient movement during scanning: misregistration introduces uninterpretable artifact, so sedation is sometimes necessary (del Rosario and Orenstein 1998).

CT and MRI are sometimes necessary for imaging extra-esophageal structures. CT interpretation is made difficult by the lack of mediastinal fat planes and IV contrast is usually necessary. Scanning is quick in the newer, multi-detector machines and sedation is rarely, if ever, needed. Care must be taken to adapt the scan parameters to the weight of the child and keep the radiation dose as low as possible. MRI excels in imaging the mediastinum and its vascular structures, where cardiac and respiratory gating ensure interpretable images without contrast. However, the long imaging time means that sedation is usually indicated.

The application of ultrasound is limited because of the location of the esophagus deep to the air-filled lungs. Gastroesophageal reflux can be imaged from the epigastric region using color-Doppler ultrasound (Jang et al. 2001). The practicality and efficacy remain to be determined, however.

8.3 Congenital Esophageal Abnormalities: Their Basis in Embryology

Pathology of the pediatric esophagus can be loosely classed as either anatomic or functional, with congenital and acquired lesions in each category. Congenital anatomic abnormalities originate in defective morphogenesis early in embryonic life (Berrocal et al. 1999; Moore 1989). Lateral tracheoesophageal folds form in the embryonic foregut during the fourth week of development. These fuse and allow the esophagus and trachea to lengthen independently. The trachea remains united with the developing pharynx cranial to the folds, through the laryngeal aditus. The esophagus is initially very short, but reaches its final length as the embryo grows. The epithelium of the esophagus proliferates and almost obliterates the lumen; recanalization occurs during the eighth week of development. Defects in the progression of fold fusion, recanalization or lengthening result in congenital anatomic abnormalities that the radiologist is usually called upon to characterize.

8.3.1 Esophageal Atresia and Tracheoesophageal Fistula

Incomplete differentiation of the presumptive trachea from the esophagus in the fourth week of embryonic development results in a spectrum of abnormalities that come to clinical attention immediately, when the infant cannot swallow oral secretions and a tube cannot be passed to the stomach. The largest group (82%) (Buonomo et al. 1998) has esophageal atresia (EA) with a distal tracheoesophageal fistula (TEF). This is visible on plain film, with an air-filled sac projecting in the neck, perhaps with a coiled tube, and air in the stomach and gut. Contrast studies are not necessary and will cause aspiration. If imaging is necessary, air insufflation of the upper sac under fluoroscopy may suffice, otherwise careful injection of a very small amount of low-osmolar water-soluble contrast may delineate the needed field. The infant's head should be elevated and the contrast quickly removed. A few babies in this spectrum (6%) have an isolated TEF (Fig 8.1). A few others not considered part of this group have laryngeal cleft, a high defect at the level of the aditus that can be confused clinically and fluoroscopically with TEF.

The surgeon may wish the radiologist to more extensively study a smaller group prior to surgery: the 10% that have atresia without TEF or with a TEF from the proximal esophageal segment. These babies will have no air distally in the GI tract. The timing of the surgery may depend on the growth of the proximal and distal esophageal segments; the smaller the distance between the ends, as demonstrated by the approximation of radio-opaque catheters under fluoroscopy (Rossi et

Fig. 8.1. Newborn male with choking and hypoxemia with feeding. Lateral view from barium swallow delineates TEF, with contrast in the trachea

al. 1998), the more successful the repair. Post-operative follow-up must be alert for early anastomotic leaks, late anastomotic strictures, and recurrent fistulas (Cumming and Williams 1996). Pneumothorax, pneumomediastinum or pleural effusion might be signs on plain films of developing complications.

All of these infants must be screened for the skeletal, renal, anal and cardiac anomalies that can coexist with EA-TEF in the VATER or VACTERL associations, in about half of the patients (Buonomo et al. 1998). Vertebral and limb abnormalities can be diagnosed by plain film, while ultrasound is the most efficient screen for the brain and heart. Anorectal malformations may be apparent clinically. Repair of the TEF may not relieve all respiratory symptoms; tracheomalacia almost always occurs when there is TEF and the residual tracheal weakness may demand its own surgical intervention.

8.3.2 Other Causes of Esophageal Narrowing

If the esophagus is intact, diagnosis may be delayed until the infant begins to exhibit feeding difficulties: food refusal, slow feeding, vomiting. Again, the spectrum of possible embryologic errors gives several differential diagnostic possibilities. The esophagus may be very short, due to defective lengthening. Plain film and barium study show the stomach in the chest (Fig. 8.2). Defective luminal recanalization can result in webs (often in mid-esophagus, but can occur at any level) or longer-segment fibromuscular stenosis, usually in the lower third of the esophagus (del Rosario and Orenstein 1998). Embryonic remnants in the esophageal wall can cause luminal narrowing. These can take the form of respiratory tract traces, such as cartilage, which will stiffen the wall and interfere with peristalsis. This can occur in conjunction with tracheoesophageal fistula and esophageal atresia, and may be overlooked due to the magnitude of these other abnormalities (Dohil and Hassall 1998).

A smoothly-curving defect in the barium-filled esophagus indicates an extraluminal mass that may be intrinsic to the esophageal wall or extrinsic to the esophagus. The esophagus is the second-most common site for duplication, after the ileum (Berrocal et al. 1999). It can form a cyst lined with secretory mucosa that causes increasing symptoms as it grows. The mucosa can also be gastric in differentiation; its presentation will be due to ulceration. The location is usually in the intrathoracic esophagus. The same type of embryologic error in the respiratory tree forms bronchogenic cysts (Nobuhara et al. 1997). In their usually central location, close to the carina, they can cause respiratory symptoms that may mask feeding problems and delay diagnosis. If complicated

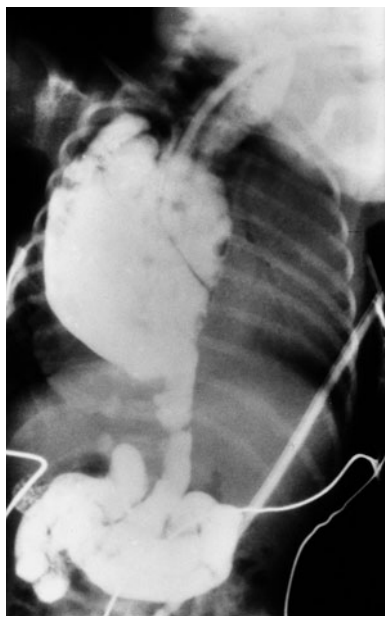

Fig. 8.2. Newborn female with cystic right chest mass on prenatal ultrasound. Barium fills the intrathoracic stomach and short esophagus

by infection, their presence may be detected by a mediastinal air-fluid level on plain film (Hedlund et al. 1998) Leiomyomas are rare smooth muscle tumors, and the esophagus is an unusual place for them, but they can occur. In children, they are more likely to be multiple or infiltrative than in adults. Leiomyomatosis is a very rare condition seen in about 5% of patients with Alport syndrome where widespread involvement of the esophagus and, occasionally, the tracheobronchial tree by smooth muscle proliferation leads to debilitating symptoms (Guest et al. 2000). Pericarinal adenopathy of sufficient magnitude in a small infant can compress the esophagus as well (Fig. 8.3).

Narrowing of the cervical esophagus has a slightly different differential list, influenced by the proximity of the branchial clefts. Cysts can form from second cleft remnants, while the third and fourth clefts can fistulize to the piriform sinuses. Cysts of the thyroid, thyroglossal duct, thymic remnants or larynx can also occur. Duplication is a rare finding in this region (Wooton-Gorges et al. 2002).

Because of the complexity of embryonic morphogenic processes, multiple errors can coexist. It behooves the radiologist to recognize patterns of malformation when they occur, but not to be a slave to them. In so doing he might reject the possibility of a certain pathology because it doesn't "fit the pattern", and thereby miss a diagnosis. For example, a child with thoracic vertebral anomalies who had other VATER/VACTERL elements excluded as an infant should not be dismissed as having behavioral problems if he presents later with feeding difficulties. Rather, a search for a neuroenteric cyst or duplication cyst should be considered, both of which can also coexist with spina bifida, hemivertebra and fusion defects (Berrocal et al. 1999). Finding one pathology should not stop the search for others. Case reports abound in the literature illustrating odd and unexpected assortments of allied anomalies: EA-TEF with esophageal stenosis (Newman and Bender 1997), TEF with web and duplication (Snyder et al. 1996), anomalous origin of a bronchus from the esophagus (Lallemand et al. 1996).

8.3.3 Vascular anomalies

Vascular rings and slings deserve special mention, as they can be definitively described radiologically and treatment planned accordingly. Like other abnormalities that narrow rather than interrupt the esophagus, these may come to clinical attention when the child's diet changes from liquid to solid. The embryonal system of parallel, interconnecting dorsal and ventral arteries normally recedes focally to leave the familiar left aortic arch and its branches. Variation in the pattern of recession can leave the fetus with arteries that cross and impinge on mediastinal structures (Strife et al. 1998). Two entities that form anatomically complete rings that encircle the trachea and esophagus and are fairly common are the double aortic arch (Fig. 8.4) and the right arch with aber-

Fig. 8.3. 6 month-old male, 2 months after a visit from foreign relatives. Coronal MRI shows compression of mediastinal structures by pericarinal and paratracheal adenopathy. The patient was treated for tuberculosis

Fig. 8.4. 5 month-old male with feeding difficulty, stridor, and distorted esophagus on barium swallow. Coronal MRI shows the trachea compressed between the double aortic arches

rant left subclavian artery and intact ductus or ligamentum (Fig. 8.5). These patients will present with respiratory and feeding difficulties though when the former is thought to cause the latter, diagnosis can be delayed. Surgical treatment is usually mandated. The arch with contralateral subclavian aberrancy, such as the very common left arch with aberrant right subclavian, will not have respiratory symptoms if the ligamentum is broken or elongated. Though a posterior defect may be seen in the barium-filled esophagus from the aberrant vessel, surgery is not usually necessary unless symptoms interfere with growth. Likewise a pulmonary sling, where the left pulmonary artery passes between the trachea and esophagus on its way to the left side from its anomalous origin on the right, may distort the esophagus without compression (Fig. 8.6); its compressive effect on the tracheobronchial tree, however, may be profound and this can secondarily interfere with feeding (Siripornpitak et al. 1997). Where a vascular anomaly is suspected, either primarily or uncovered on barium esophagram, MRI can definitively delineate it and show its effect on mediastinal structures; angiography is contraindicated.

Fig. 8.5. 2 month-old female with wheezing and regurgitation, and right aortic arch on chest x-ray. Lateral view of barium swallow shows prominent posterior defect. MRI (not shown) confirmed vascular ring with right arch, aberrant left subclavian artery and intact ligamentum

8.4 Congenital Functional Abnormalities: The Interplay of Anatomy and Maturation

8.4.1 Dysphagia

Swallowing demands a certain level of central nervous system (CNS) maturation and function. Musculoskeletal structures in the face and neck must act in flawless concert to safely deliver ingested food past the larynx and into the esophagus. Similar functions are brought into play to protect the airway during reflux or vomiting. Swallows can be observed in the 12-week fetus, and passage of amniotic fluid through the GI tract is essential for its growth and development. However, the oral phase of swallow requires skills that come with later fetal maturity (Derkay and Schechter 1998). Premature infants' suckle behavior is characterized by short bursts of quick jaw movements and tongue compressions. This persists in the mature infant as nonnutritive sucking, an important behavioral adjunct to successful feeding. However, premature infants cannot reliably mount the negative intraoral pressure required for extraction of fluid from the nipple and formation of a swallow bolus. Thus, an esophagram

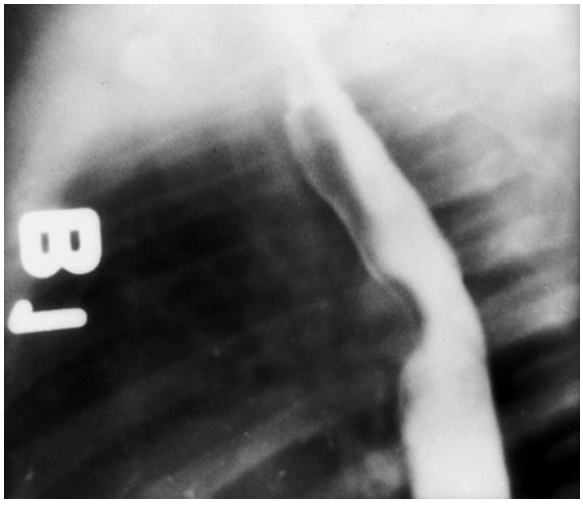

Fig. 8.6. 1 month-old male with wheezing. Chest radiograph (not shown) was remarkable for asymmetric hyperinflation on the right. Lateral view of barium swallow shows anterior defect in the contrast column, compatible with pulmonary sling. Confirmed by MRI (not shown)

on such an infant may show fluid loss from the mouth, uncontrolled entry of fluid to the pharynx, nasopharyngeal reflux, penetration of fluid to the larynx and trachea, lack of cough response, poor coordination of breathing and swallowing. All of these are cause for great concern when they occur in the term or near-

term infant, but may prompt only watchful waiting and cautious re-evaluation at a later time in a baby who could be 25-32 weeks post-conception age at the time of the first exam. However, in both groups the finding of contrast in the trachea should be scrutinized carefully to exclude the possibility of TEF or laryngeal cleft. Using a small, thin-walled nipple and observing swallows at the start of the exam before the patient tires help the premature infant show his best abilities.

Infants and children with CNS dysfunction secondary to primary brain dysplasia (due to prenatal infection or vascular events, or to chromosomal abnormality [Eicher et al. 2000]) and perinatal hypoxemia or hemorrhage form a large and growing population of patients with impaired swallowing. Loss of swallow skills from head trauma, malignancy, or degenerative disorders adds these patients to this group as well. Occasionally, progressive dysphagia is the presenting symptom that leads to diagnosis (Elta et al. 1996), or it may be only one of many problems. Advances in salvage and supportive care have made the basic ability to be fed the remaining determinant of survival in this population. It is the job of the radiologist, often in tandem with a feeding therapist, to determine a patient's level of function, whether there are any anatomic impediments to safe feeding, and what food textures can be fed (Gisel et al. 1998). The esophagram is often used to elucidate the basic anatomic framework and exclude obstruction, followed by a modified barium swallow concentrating on the swallow mechanism (Mercado-Deane et al. 2001).

8.4.2 Gastroesophageal Reflux

Gastroesophageal reflux (GER) is near-universal in infants, decreasing in frequency with growth and maturation. It still occurs in normal children as well. It becomes pathologic when associated with pain, respiratory symptoms, or dysphagia. Transient lower esophageal sphincter relaxation (TLESR) has been implicated in allowing reflux episodes to occur but many factors contribute to the transition of reflux from a benign to a malignant classification (del Rosario and Orenstein 1998). One such factor is decreased esophageal clearance, from supine positioning, dysmotility or infrequent swallowing. The presence of a nasogastric feeding tube can also impede acid clearance, though the number of reflux episodes is unchanged; a large tube can convert a non-refluxer to a refluxer, however (Noviski et al. 1999). Straining, a favorite maneuver to provoke reflux in adults during a fluoroscopic examination, does not do the same in children, unless it happens to coincide with a TLESR (Kawahara et al. 2001). High gastric volume (due to large feeds or delayed gastric emptying) and increased osmolality in gastric contents are associated with more and longer periods of lowered LES pressure (Salvia et al. 2001). Hiatus hernia promotes reflux because of the loss of diaphragmatic hiatal support for the LES.

The most important morbidity associated with GER is aspiration. The effects of gastric acid on the tracheobronchial tree visible on plain film may be subtle hyperinflation due to vagal-mediated bronchospasm or flagrant peribronchial inflammation, atelectasis, or pneumonia. In the child who is usually in a supine position, the upper lobes, particularly on the right, may be more involved; a child who is more erect will deposit the aspirate into the lower lobes. Even aspiration of saliva, if it occurs chronically, is far from benign. Distinguishing between salivary and gastric aspiration can be important for therapy, and in cases where GER studies fail to show expected reflux, nuclear medicine salivagram can be useful (Cook et al. 1997).

8.4.3 Achalasia

Successful passage of a food bolus through the esophagus depends on sequential relaxation and closing of the upper and lower sphincters that divide the esophagus from the pharynx above and the stomach below. Achalasia of the LES is unusual in children, comprising only about 5% of the total cases (Buonomo et al. 1998). In older children, (mean age of onset being about 9 years) the radiologic appearance is like that in adults, with a distended, static esophagus terminating in a "beak" at the LES. In younger patients, the distension is less evident. The classic therapy is surgical myotomy; in recent years hydrostatic balloon dilatation has become popular (Upandhyaya et al. 2002). However, in all patients there is an underlying dysmotility that affects the entire esophagus, such that relief of the LES obstruction does not restore normal esophageal function. Follow-up with barium and radionuclide swallows in a series of patients showed persistent esophageal dilatation and contrast retention despite an open cardia (Chawda et al. 1998). This paper also indicated that radionuclide studies could substitute for barium swallows for functional follow-up, at a lower radiation dose.

Cricopharyngeal achalasia is still more rare, with scattered cases reported (De Caluwe et al. 1999).

The characteristic cricopharyngeal bar (a posterior indentation in the barium column during swallow) can be seen intermittently in normal patients, but true achalasia of the upper esophageal sphincter (UES) results in obstruction to passage of the bolus, with discoordinate swallowing, cough, nasopharyngeal reflux, and aspiration. Arnold-Chiari malformation can be a cause, and should be excluded (del Rosario and Orenstein 1998). The same treatments used for LES achalasia can apply here, but in some cases conservative treatment with gavage feeding showed slow return to normal UES function.

Special mention should be made of children with Down syndrome. They are disproportionately represented among patients with congenital GI abnormalities, including esophageal webs, TEF/EA, duodenal atresia and Hirschsprung disease. They also have a high rate of esophageal dysmotility and LES achalasia, up to 30% in one study (Zárate et al. 2001).

8.5 Acquired Anatomic Abnormalities: Idiopathic and Iatrogenic

8.5.1 Foreign Body Ingestion

The most frequent acquired pathology among children is the ingested foreign body. The bounty of case reports in the literature attests to the child's endless ingenuity in this regard. Coins are the most frequently swallowed object (Harned et al. 1997); the local demographics will determine if the second-most-frequent items are fish bones or other metal objects such as batteries or keys (Cheng and Tam 1999; Loh et al. 2000). Diagnosis is straightforward in the case of a radio-opaque item: lateral film of the lower face and neck and an AP film of the chest and abdomen to cover the entire GI tract from nose to anus will locate it. The lateral examination is recommended because of the young child's ability to habituate to foreign bodies in the pharynx and nasopharynx, which could be missed on chest x-ray (Fig. 8.7). Diagnosis of a bone ingestion is more difficult, but one study confirmed the plain film appearance in 26% (Cheng and Tam 1999). If the foreign body passes the UES, it will tend to impact at points where the esophageal lumen narrows: the thoracic inlet, the carina, and the LES. There is no consensus in the literature about predicting passage based on size and location at diagnosis, but there is unanimity in the assertion that all esophageal foreign bodies should be removed before 24 hours. The risk of inflammation and perforation is high and containment is poor, as the esophagus has no serosa.

Retrieval method for the foreign body varies with the institution, the kind of object, and how long it has been impacted. Coins are most frequently removed under fluoroscopy using a Foley catheter (Harned et al. 1997). Sharp, small or multiple objects should be removed endoscopically or surgically, as should coins where the radiographic appearance suggests tracheal compression or edema, or possible perforation (pneumomediastinum, pleural effusion) (Kaye and Towbin 1996). Button batteries, such as for cameras or hearing aids, should be immediately removed as they cause chemical damage to the mucosa on contact and are a prominent cause of perforation (Samad et al. 1999).

The consequences of delayed removal are grave. High impactions at the UES can present as wheezing, cough and vocal cord paralysis (Virgilis et al. 2001). Sharp objects such as fish bones can lacerate the mucosa leaving hematomas (a minor complication is this series) or perforate (7% of patients), causing mediastinitis, retropharyngeal abscess, and aorto-esophageal fistula (Loh et al. 2000). Pieces of plastic often have sharp edges and can erode and perforate (Fig. 8.8). The level of perforation does not necessarily determine the nature of the complication, as the mediastinitis that inevitably follows can transmit infection to distant sites, such as an intervertebral disc (Fonga-Djimi et al. 1996). Because it is smooth, a coin in the esophagus can be initially asymptomatic. However with time, it too can erode and cause infection or per-

Fig. 8.7. 16 month-old female with two-week history of food refusal. AP scout radiograph for modified barium swallow. Hoop earring is seen impacted at the thoracic inlet. Patient transferred to the endoscopy service

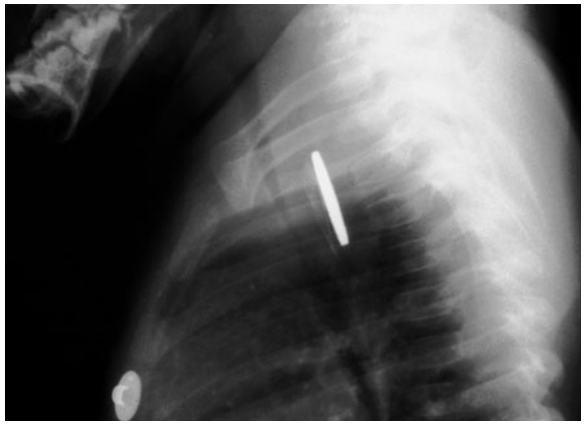

Fig. 8.8. 11 month-old female with drooling, wheezing, irritability and food refusal. Lateral chest radiograph. Coin in the esophagus is seen on edge. Just anterior to it is the faint outline of a piece of broken plastic. The trachea is narrowed, indicating mediastinitis. This patient is *not* a candidate for extraction by Foley catheter because of the multiplicity, the sharp edges, and the complications

foration; one case report detailed a brain abscess that followed delayed extraction (Louie et al. 2000).

8.5.2 Iatrogenic Foreign Bodies

Nasogastric intubation is one of the most frequent hospital interventions though inadvertent perforations rarely occur. The neonate is most frequently affected, probably because of the small diameter of the tract relative to the size of the tube, and the fact that the tube must pass through an even narrower lumen at the UES. If the UES is closed because of spasm, the tube can perforate the pharynx. Films at that point might be reminiscent of esophageal atresia, with the tube curling in the neck. Air in the mediastinum, expanded retropharyngeal soft tissues and pleural effusion will help alert the radiologist to the correct diagnosis. Cautious administration of water-soluble contrast may show a pseudodiverticulum or a free perforation (Sarin et al. 2000).

Thoracostomy tubes are placed to drain air or fluid from the pleural space. There is a tendency for the operator to place the tube as far medially as possible to ensure that it does not become dislodged. However, should the tube rest against mediastinal structures such as the esophagus, pressure necrosis can result (Cairns et al. 1999). When percutaneous gastrostomy tubes are changed, the internal rubber bumper is often left in the stomach to pass in the stool. Occasionally, these can cause gastric outlet obstruction. In one case, the remnant migrated into the distal esophagus and impacted where, despite its being soft and malleable, it ulcerated the mucosa and caused scarring (Nowicki et al. 1996).

8.5.3 Mucosal Destruction

Widespread trauma to the esophageal mucosa can result in perforation acutely, or eventually lead to scarring and narrowing. The cause can be chemical burns, infection or inflammation. Corrosive ingestions do the most damage when the agent is an alkali, such as drain cleaner. Burns are found most usually in the mouth, with fewer patients exhibiting pharyngeal involvement and still fewer with esophageal burns. In one series, 29% had complications and about half of these required esophageal replacement (de Jong et al. 2001). Contrast esophagram evaluation is generally delayed until after the initial inflammation subsides; if done earlier, it tends to underestimate the extent of damage. It is most valuable in assessing esophageal function in cases of moderate burns and gauging the extent of strictures and the results of dilatation. Immunosuppressed patients with oral candidiasis can develop candidal esophagitis. Graft vs. host disease, a feature of the same population, can behave similarly. Where the mucosa is too friable for endoscopy, contrast swallow can help assess the extent of ulceration, response to therapy and the occurrence of strictures. In advanced, invasive candidiasis cases perforation can occur, necessitating esophageal replacement (Gaissert et al. 1999). Stevens-Johnson syndrome, or toxic epidermal necrolysis, is characterized by skin and mucosal lesions, precipitated by certain drugs or infectious agents. Oral lesions are frequent (60-80% of patients) and their severity can mask esophageal involvement. Strictures are not common sequelae, however (Lamireau et al. 2001).

Reflux esophagitis has several recognized complications. Barrett's esophagus, where a specialized epithelium known to be preneoplastic is found in an esophageal segment of variable length, is found particularly in patients predisposed to severe GER: those with severe neurologic impairment who are mainly recumbent, those with chronic lung disease such as cystic fibrosis who cough and have pulmonary toilet in the head-down position, those with primary esophageal dysmotility or hiatus hernia (Hassall 1997). Mucosal irregularity, ulceration, and narrowing can be seen on contrast study. Schatzki rings, less familiar in children than in adults, are found in a similar

group of patients (Buckley et al. 1998). Esophagitis can have a predictable outcome – multiple mucosal rings – but an unusual cause: eosinophilia with food allergy, not reflux (Siafakas et al. 2000).

8.5.4 Miscellaneous Causes of Esophageal Dysfunction

Esophageal perforation due to blunt chest trauma is a familiar scenario for an adult in a motor vehicle accident, but very rare in a child. Diagnosis can be delayed unless there is a high index of suspicion (Sartorelli et al. 1999). Iatrogenic perforation during stricture dilatation is more common, 21% in one series (Kang et al. 1998) where balloon dilatation was used. Anastomotic (post EA repair) and peptic strictures reacted the same way to dilatation. Another series (Panieri et al. 1996) documented less that 1% perforations with bougie and olive dilators. Contrast swallows in these series showed abnormality ranging from mucosal disruption to frank perforation or fistula.

Nissen fundoplication is the surgical procedure of choice to control GER. It has its own complications, however, which can adversely affect the patient's ability to eat (Trinh and Benson 1997). Tightening the gastroesophageal junction is a delicate balance: tight enough to prevent reflux but loose enough to permit bolus passage. Patients who have had an EA-TEF repaired already suffer from dysmotility of the esophagus below the anastomosis. This promotes reflux and usually is the reason for the Nissen. However, the ability to propel the bolus may then be completely compromised. Fundoplications can completely break down, but more often a large paraesophageal hernia forms that extends into the chest through the loosened hiatus. This can compress the distal esophagus still further and impede bolus passage. Many Nissen patients are not fed by mouth but by gastrostomy tube and it is tempting, when asked to look for recurrent reflux to explain the patient's vomiting, to only inject the tube. However, giving just a few cc's of barium by mouth can find the possible distal esophageal obstruction and show the distended esophagus filled with secretions that the patient regurgitates.

Two final acquired complications can narrow the esophagus. In one, an arterial switch procedure puts the aorta into position to compress the esophagus (McElhinney et al. 1998). The other represents the end stage of any of a number of diseases that cause portal hypertension (Buonomo et al. 1998). Extrahepatic portal obstruction is much more common in children. Varices form around the distal esophagus, causing vermiform defects in the margins of the contrast column.

8.6 Acquired Functional Abnormalities: The Diagnosis of Exclusion

Difficult feeding or food refusal suspected to be psychogenic in origin is a frequent reason for ordering a modified barium swallow prior to instituting behavioral therapy. There are some historical cues that can support this request: early history of prematurity or surgical procedures that interfered with the evolution of feeding skills, or bizarre dietary choices that have no logic. However, all of the forgoing chapter has attempted to show that there can be a compelling anatomic or functional pathology to explain the symptoms, and that it is the radiologist's job to find it. History clues, such as suddenness of onset, wheezing, coughing, irritability, and taking liquids but refusing solids all point toward a physical cause. A formal upper GI exam should precede any modified swallow. It is a small, "low-tech" effort that can reap impressive results. Once the radiologist is satisfied that the child is normal anatomically and functionally, a psychogenic diagnosis might be entertained.

References

Berrocal T, Torres I, Gutierrez J, Prieto C, del Hoyo ML, Lamas M (1999) Congenital anomalies of the upper gastrointestinal tract. Radiographics 19:855–872

Buckley K, Buonomo C, Husain K, Nurko S (1998) Schatzki ring in children and young adults: clinical and radiologic findings. Pediatr Radiol 28:884–886

Buonomo C, Taylor GA, Share JC, Kirks DR (1998) Gastrointestinal tract. In: Kirks DR, Griscome NT (eds) Practical pediatric radiology. Lippincott-Raven, Philadelphia, New York, pp 821–1008

Cairns PA, McClure BG, Halliday HL, McReid M (1999) Unusual site for esophageal perforation in an extremely low birth weight infant. Eur J Pediatr (1999) 158:152–153

Chawda SJ, Watura R, Adams H, Smith PM (1998) A comparison of barium swallow and erect esophageal transit scintigraphy following balloon dilation for achalasia. Dis Esophagus 11:181–188

Cheng W, Tam PKH (1999) Foreign-body ingestion in children: experience with 1,265 cases. J Pediatr Surg 34(10):1472–1476

Cook SP, Lawless S, Mandell GA, Reilly JS (1997) The use of the salivagram in the evaluation of severe and chronic aspiration. Int J Pediatr Otorhinolaryngol 41:353–361

Cumming WA, Williams JL (1996) Neonatal gastrointestinal imaging. Clin Perinatol 23(2):387–407

De Caluwe D, Nassogne MC, Reding R, de Ville de Goyet J, Clapuyt Ph., Otte JB (1999) Cricopharyngeal achalasia: case reports and review of the literature. Eur J Pediatr Surg 9:109–112

De Jong AL, Macdonald R, Ein S, Forte V, Turner A (2001) Corrosive esophagitis in children: a 30-year review. Int J Pediatr Otorhinolaryngol 57:203–211

Del Rosario JF, Orenstein SR (1998) Common pediatric esophageal disorders. Gastroenterologist 6(2):104–121

Derkay CS, Schechter GL (1998) Anatomy and physiology of pediatric swallowing disorders . Otolaryngol Clin North Am 31(3):397–404

Dohil R, Hassall E (1998) Esophageal stenosis in children. Gastrointest Endosc Clin N Am 8(2):369–375

Eicher PS, McDonald-McGinn DM, Fox CA, Driscoll DA, Emanuel BS, Zackia EH (2000) Dysphagia in children with a 22q11.2 deletion: Unusual pattern found on modified barium swallow. J Pediatr 137(2): 158–164

Elta GH, Caldwell CA, Nostrant TT (1996) Esophageal dysphagia as the sole symptom in type 1 Chiari malformation. Dig Dis Sci 41(3):512–515

Fonga-Djimi H, Leclerc F, Martinot A, Hue V, Fourier C, Deschildre A, Flurin V (1996) Spondylodiscitis and mediastinitis after esophageal perforation owing to a swallowed radiolucent foreign body. J Pediatr Surg 31(5):698–700

Gaissert HA, Breuer CK, Weissburg A, Mermel L (1999) Surgical management of necrotizing Candida esophagitis. Ann Thorac Surg 67(1):231–3

Gisel EG, Birnbaum R, Schwartz S (1998) Feeding impairments in children: diagnosis and effective intervention. Int J Orofacial Myology 24:27–33

Guest AR, Strouse PJ, Hiew CC, Arca M (2000) Progressive esophageal leiomyomatosis with respiratory compromise. Pediatr Radiol 30:247–250

Harned RK, Strain JD, Hay TC, Douglas MR (1997) Esophageal foreign bodies: safety and efficacy of foley catheter extraction of coins. Am J Roentgenol 168:443–446

Hassall E (1997) Comorbidities in childhood Barrett's esophagus. J Pediatr Gastroenterol Nutr 25:255–260

Hedlund GL, Griscome NT, Cleveland RH, Kirks DR (1998) Respiratory system. In: Kirks DR, Griscome NT (eds) Practical pediatric radiology. Lippincott-Raven, Philadelphia, New York, pp 619–820

Hernandez RJ, Goodsitt MM (1996) Reduction of radiation dose in pediatric patients using pulsed fluoroscopy. AJR 167:1247–1253

Jang HS, Lee JS, Lim GY, Choi BG, Choi GH, Park SH (2001) Correlation of color Doppler sonographic findings with pH measurements in gastroesophageal reflux in children. J Clin Ultrasound 29(4):212–216

Kang SG, Song HY, Lim MK, Yoon HK, Goo DE, Sung KB (1998) Esophageal rupture during balloon dilation of strictures of benign or malignant causes: prevalence and clinical importance. Radiology 209:741–746

Kawahara H, Dent J, Chir B, Davidson G, Okada A (2001) Relationship between straining, transient lower esophageal sphincter relaxation, and gastroesophageal reflux in children. Am J Gastroenterol 96(7):2019–2025

Kaye RD, Towbin RB (1996) Interventional procedures in the gastrointestinal tract in children. Radiol Clin North Am 34(4):903–917

Lallemand D, Quignodon JF, Courtel JV (1996) The anomalous origin of bronchus from the esophagus: report of three cases. Pediatr Radiol 26:179–182

Lamireau T, Leauté -Labrèze C, Le Bail B, Taieb A (2001) Esophageal involvement in Stevens-Johnson syndrome. Endoscopy 33(6):550–553

Loh KS, Tan LKS, Smith JD, Yeoh KH, Dong F (2000) Complications of foreign bodies in the esophagus. Otolaryngol Head Neck Surg 123(5):613–616

Louie JP, Osterhoudt KC, Christian CW (2000) Brain abscess following delayed endoscopic removal of an initially asymptomatic esophageal coin. Pediatr Emerg Care 16(2):102–105

McElhinney DB, Reddy VM, Reddy GP, Higgins CB, Hanley FL (1998) Esophageal compression by aorta after arterial switch. Ann Thorac Surg 65:246–248

Mercado-Deane MG, Burton EM, Harlow SA, Glover AS, Deane DA, Guill MF, Hudson V (2001) Swallowing dysfunction in infants less than 1 year of age. Pediatr Radiol 31:423–428

Moore, K L (1989) Before we are born: basic embryology and birth defects, 3rd edn. Philadelphia, W. B. Saunders Company, pp 159–161

Newman B, Bender TM (1997) Esophageal atresia/tracheoesophageal fistula and associated congenital esophageal stenosis. Pediatr Radiol 27:530–534

Nobuhara KK, Gorski YC, La Quaglia MP, Shamberger RC (1997) Bronchogenic cysts and esophageal duplications: common origins and treatment. J of Pediatr Surg 32(10): 1408–1413

Noviski N, Yehuda YB, Serour F, Gorenstein A, Mandelberg A (1999) Does the size of nasogastric tubes affect gastroesophageal reflux in children? J Pediatr Gastroenterol Nutr 29(4):448–451

Nowicki MJ, Johnson ND, Rudolph CD (1996) Esophageal stricture caused by a retained percutaneous gastrostomy tube remnant. J Pediatr Gastroentero Nutr 22(2):208–211

Panieri E, Millar AJW, Rode H, Brown RA, Cywes S (1996) Iatrogenic esophageal perforation in children: patterns of injury, presentation, management, and outcome. J Pediatr Surg 31(7):890–895

Rossi C, Domini M, Aquino A, Persico, Lelli-Chiesa P (1998) A simple and safe method to visualize the inferior pouch in esophageal atresia without fistula. Pediatr Surg Int 13: 535–536

Salvia G, De Vizia B, Manguso F, Iula VD, Terrin G, Spadaro R, Russo G, Cucchiara S (2001) Effects of intragastric volume and osmolality on mechanisms of gastroesophageal reflux in children with gastroesophageal reflux disease. Am J Gastroenterol 96(6):1725–1732

Samad L, Ali M, Ramzi H (1999) Button battery ingestion: hazards of esophageal impaction. J Pediatr Surg 34(10): 1527–1531

Sarin YK, Goel D, Mathur NB, Maria A (2000) Neonatal pharyngeal pseudo-diverticulum. Indian Pediatr 37:1134–1137

Sartorelli KH, McBride WJ, Vane DW (1999) Perforation of the intrathoracic esophagus from blunt trauma in a child: case report and review of the literature. J Pediatr Surg 34(3): 495–497

Siafakas CG, Ryan CK, Brown MR, Miller TL (2000) Multiple esophageal rings: an association with eosinophilic esophagitis. Am J Gastroenterol 95(6):1572–1575

Siripornpitak S, Reddy GP, Schwitter J, Higgins CB (1997) Pulmonary artery sling: anatomical and functional evaluation by MRI. J Comput Assist Tomogr 21(5):766–768

Snyder CL, Bickler SW, Gittes GK, Ramachandran V, Ashcraft KW (1996) Esophageal duplication cyst with esophageal web and tracheoesophageal fistula. J Pediatr Surg 31(7):968–969

Strife JL, Bisset III GS, Burrows PE (1998) Cardiovascular system. In: Kirks DR, Griscome NT (eds) Practical pediatric radiology. Lippincott-Raven, Philadelphia, New York, pp 511–618

Trinh TD, Benson JE (1997) Fluoroscopic diagnosis of complications after Nissen antireflux fundoplication in children. Am J Roetgenol 169(4):1023–1028

Upadhyaya M, Fataar S, Sajwany MJ (2002) Achalasia of the cardia: experience with hydrostatic balloon dilation in children. Pediatr Radiol 32:409–412

Vazquez JL, Buonomo C (1999) Feeding difficulties in the first days of life: findings on upper gastrointestinal series and the role of the videofluoroscopic swallowing study. Pediatr Radiol 29:894–896

Virgilis D, Weinberger JM, Fisher D, Goldberg S, Picard E, Kerem E (2001) Vocal cord paralysis secondary to impacted esophageal foreign bodies in young children. Pediatrics 107(6):e101

Wootton-Gorges SL, Eckel GM, Poulos ND, Kappler S, Milstein JM (2002) Duplication of the cervical esophagus: a case report and review of the literature. Pediatr Radiol 32:533–535

Zárate N, Mearin F, Hidalgo A, Malagelada J-R (2001) Prospective evaluation of esophageal motor dysfunction in Down's syndrome. Am J Gastroenterol 96(6):1718–1724

9 The Geriatric Pharynx and Esophagus

Olle Ekberg

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9.1 Introduction

There are several age-related alterations in oral, pharyngeal and esophageal morphology and function. These variations in the healthy elderly (primary ageing) must be taken into account during clinical and radiological evaluation. In fact, theses changes do not normally impair the swallowing process and are thereby not symptomatic. However, when disease processes (secondary ageing) add to primary ageing they may result in significant impairment.

Deglutition disorders are common and costly in the elderly. The actual prevalence and natural history of such dysphagia is not very well known. It is more commonly detected in the hospitalised and institutionalised elderly than in individuals who live in their own homes. The prevalence of dysphagia in hospitals and nursing homes with predominantly an elderly population has revealed a prevalence of dysphagia in up to 50% of the population (Groher and Bukatman 1986). It is also much more common in the very old (over the age of 85) than in any other group. Neurologic diseases such as stroke, dementia and Parkinson's disease are common in these populations. The central nervous system regulates oropharyngeal function in an integrated complex sensory and motor activity pattern. Especially the co-ordination between the oral and pharyngeal stage, i.e. between a voluntary and an automatic function, is vulnerable. In the elderly this coordination is frequently impaired.

Many elderly people with dysphagia seem to be in reasonably good health, not frail or bedridden. They do not seem to suffer from clinically apparent neurologic disease. Oropharyngeal impairment is usually insidious and chronic in these individuals, although acute, transient cases do occur. A rational approach to understanding deglutition in the aged requires that we differentiate expected, if not clearly predictable, senescent changes (primary ageing) from those caused by disease (secondary ageing). Unfortunately, subclinical pathology, especially cerebral vascular disease, makes this distinction difficult.

From other organ system we know that there is roughly a 1% yearly decline in function beginning at age 30. Overall ageing is a progressive generalised impairment of function resulting in a loss of adaptive response to stress and in a growing risk of age-associated disease (Kirkwood 2000).

The morphodynamics of deglutition can be measured in terms of timing, movement of anatomical structures, pressure generation and bolus movements (Tracy et al. 1989; Sonies et al. 1988; Shaw et al. 1990; Shaker et al. 1990). Senescent changes in oropharyngeal function can be characterised as "less efficient". However, the relationship between symptoms and morphodynamic abnormalities, especially in a context of compensation/decompensation is very complex (Jones and Donner 1991).

One important feature of ageing is an inability to adapt to stress and the videofluorographic examination is certainly a stressful situation. Abnormalities, or differences from younger, presumably normal individuals, may induce alterations that reflect senescent

O. Ekberg, MD
Professor, Department of Diagnostic Radiology, Malmö University Hospital, 205 02 Malmö, Sweden

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decline in function. Similarly, the examination itself may induce decompensation in those with existing dysfunction due to known disease and minor abnormalities may become major.

9.1.1 The Ageing Brain

In the central nervous system various alterations of the cyto-architecture occurs in ageing. For example, motor neuron counts in the spinal cord show that the number of cells declines by approximately 200 neurons per segment per decade (Schoenen 1991). In a study that counted the pigmented neurons in the locus coeruleus in the brainstem found a decline averaging 2000 cells per decade after the age of 60 (Vijayashankar and Brody 1979). Cell counts in various glossopharyngeal and vagus nuclei within the brainstem are not available in humans but a similar decline is likely.

9.1.2 Primary Ageing

The oral cavity is undergoing important changes with age. Such changes include an increase in the amount of connective tissue in the tongue, loss of dentition and reduced masticatory strength (Logemann 1990). In the pharynx it has been shown that the anterior elevation of the larynx is less pronounced in the elderly and that the pharyngeal swallowing phase is significantly slowed down (Sonies et al. 1988). The pharyngeal peristaltic motion has been shown to be slowed down above the age of 60 (Tracy et al. 1989). However, another study showed that this is not the case but that there is instead a wider intrapersonal variability in the aged (Borgström and Ekberg 1988a). However, there is no significant change in pharyngeal peak pressure, duration or in the rate of propagation of contraction (Robbins et al. 1992). Healthy elderly subjects therefore do not have any residual accumulation in the pharynx after swallowing.

9.2 The Oral Stage

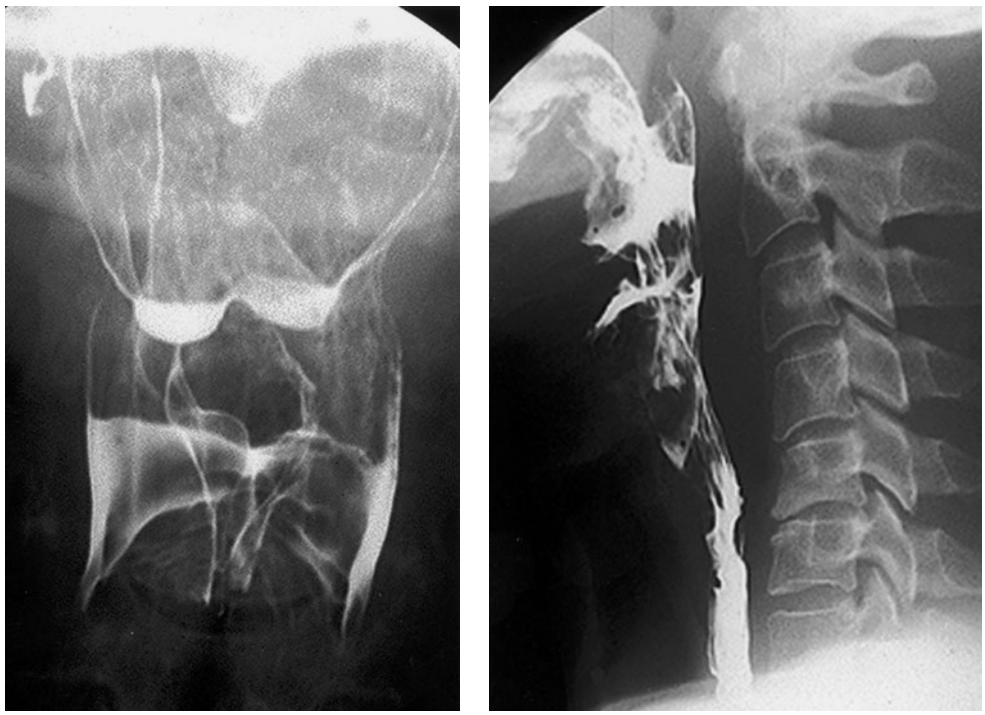
It has been shown that pressure that is generated during swallowing by the tongue does not change in elderly compared to young individuals. However, when individuals are asked to apply maximal strength the pressure recording increases considerably in young, while in the elderly such reserve capacity does not seem to exist. Others have shown that lingual peristaltic pressure decreased with age (Shaker et al. 1988)

9.3 The Pharyngeal Stage

9.3.1 Misdirected Swallowing

Bolus misdirection into the airways is the most significant event observed during videofluoroscopy. The timing and level of bolus misdirection are important observations, but again, may be extremely variable (Dodds et al. 1990; Logemann 1983; Groher 1983). In the elderly, it is not uncommon to see ingestion of a too large a bolus volume or rapid ingestion rate during uncontrolled administrations. This results in disruption of sequencing between bolus ingestion, delivery, propulsion and laryngeal closure. Some patients aspirate only on the first liquid barium administration, others appear relatively normal until a small amount penetrates and then grossly deteriorate and aspirate before, during and after some of the subsequent swallows. Particularly in the elderly bolus misdirection most often results from oral stage dysfunction even when it occurs during the pharyngeal stage.

Another common finding in the elderly is failure of containment during ingestion, oral processing of bolus holding. In these individuals the glossopalatal seal is inadequate. Lingual movements may be dyskinetic or disorganised and disrupt the glossopalatal seal. Alternatively there may be no oromotor activity attempting to contain the bolus once it is ingested. The third oral stage cause of misdirection is transitional dissociation. This means that the bolus is positioned at the wrong place at the wrong time. Dissociation has been defined in Chap. 4. In young individuals the hyoid bone starts to move anteriorly (from its posterior position) before the apex of the bolus passes the level of the faucial isthmus as it can be seen in a true lateral projection during swallowing. With increasing age the start of this anterior hyoid bone movement is delayed. It is common in elderly (over 75 years of age) to observe a delay of the anterior movement of the hyoid bone for more than 0.5 s after the apex of the bolus passed the faucial isthmus (Feinberg and Ekberg 1991). In that study using the previously



a b

Fig. 9.1a,b. Pharyngeal dysfunction is common in the elderly. Weak musculature leads to impaired clearance of the pharynx. This is mostly related to defective elevation of the pharynx. In this patient there is pooling of contrast medium in the vallecula and piriform sinus bilaterally. Contrast medium has reached into the laryngeal vestibule and is also seen between the arytenoids

outlined pattern analysis it was found that aspiration was due to oral stage dysfunction in 50%, pharyngeal dysfunction in 30% and combined dysfunction in 20%. The most common oral stage abnormality was failure of containment. Transitional dissociation was seen in many patients. Almost as common was large bolus ingestion or rapid ingestion rate. The most common pharyngeal stage abnormality was incomplete transportation, i.e. retention. A mere defective laryngeal closure was seen less frequently (Feinberg and Ekberg 1991). There is, however, no significant relationship between a patient's specific disease, and the pattern of abnormalities during barium swallow. This means that the observed dysfunctions are non-specific in terms of their aetiology. The high frequency of oral dysfunction indicates that oral stage abnormalities must be routinely looked for during videofluoroscopy.

The presence of anterior osteophytes larger than 10 mm that impinge on the pharynx may explain aspiration in dysphagic patients (Strasser et al. 2000). Coexisting clinical conditions and diseases such as stroke and partial laryngeal resection increases the risk of aspiration in patients with smaller osteophytes of the cervical spine.

Pneumonia is common in the elderly and it may be due to defective closure of the airways during swallowing, i.e. penetration/aspiration. However, the cause and effect relationship is complex and few studies have addressed this accordingly (Doggett et al. 2001; Feinberg et al. 1996). In fact, the most plausible explanation is that infected saliva reaches the lower airways by route of the larynx and causes the infection (Langmore et al. 1998).

9.3.2 Pharyngeal Constrictors

There are conflicting opinions concerning the effect of aging on deglutitive pharyngeal pressures. While one study showed that there was no pressure difference (Robbins et al. 1992), other studies have shown differences (Tracy et al. 1989; Perlman et al. 1993; Shaker et al. 1993). However, there is a multitude of methodological differences that makes comparison difficult. In fact, one of the studies (Shaker et al. 1990) showed that the peristaltic pressure wave, amplitude and duration was significantly greater in the elderly compared to the young. Such alteration

with age, alterations that actually could be seen as improvements, may actually be regarded as compensatory responses to, for instance, reduced cross sectional area of the deglutitive pharyngoesophageal segment opening in the elderly (Shaw et al. 1990). A study supporting this theory has been presented by Shaker (Shaker 1993). In this study he found that intrabolus pressure in the pharynx was significantly higher in elderly compared to young patients, both on swallowing in upright and supine positions and both with liquid and mashed potatoes. This again may indicate that there is lack of distension of the PES in the elderly.

9.3.3 The Pharyngoesophageal Segment

The pharyngoesophageal sphincter pressure has also been studied in elderly compared to younger subjects. Cook et al. (1989) did not find significant age-related changes in resting PES pressure measured with a sleeve device; however, he only studied individuals under 55 years of age. Wilson et al. (1989) made the same observation in healthy subjects under 62 years of age. On the other hand, Fulp et al. (1990) were able to show that normal elderly subjects over 62 years of age have lower resting PES pressure than younger controls. Shaker et al. (1993) studied the effect of ageing (70 years and over) on the resting PES pressure and its response to esophageal air and balloon distension. The results of this study indicate that aging significantly reduces the resting PES pressure. However, this latter study (Shaker et al. 1990, 1993) also showed that there was normal PES pressure responses to swallowing and esophageal distension by air and balloon in the elderly. Therefore, the protective role of the PES against pharyngeal reflux of gastric acid is preserved in the elderly.

Another study by Kendall and Leonard (2001) has shown that there is pharyngeal weakness in the elderly dysphagic patient. They also concluded that poor pharyngeal constrictions suggestive of pharyngeal weakness contributed to 75% of cases of aspiration in their study. Feeding and respiratory pattern was studied in normal older people (Hirst et al. 2002). Hirst and colleagues found a fairly stable pattern. It has been shown that particularly in the elderly incoordination commonly leads to aspiration (Nilsson et al. 1997). Solid aspirators also have a lower oxygen saturation level compared to normals (Colodny 2001).

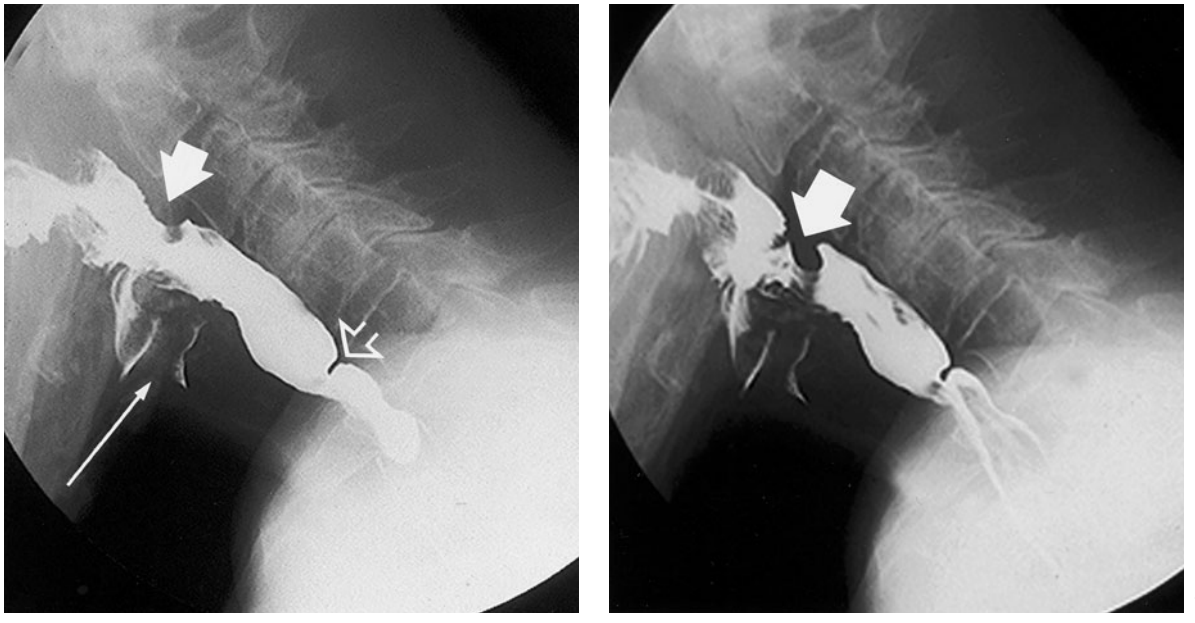


Fig. 9.2a,b. Barium swallow in a 79-year-old woman with dysphagia. She coughs during eating. Compensation is often difficult to detect on barium swallow. This patient has increased activity in the middle pharyngeal constrictor (*broad arrow*). This is likely to be due to a weak tongue base pressure. In this patient there is also misdirected swallowing. Barium has reached through the vocal cords (*arrow*) into the subglottic area. There is also an incoordination of the opening of the pharyngoesophageal segment seen as an indentation of the cricopharyngeal muscle (*open arrow*). **a** Midpharyngeal stage of swallow. **b** Two eighths of a second after (**a**)

Fig. 9.3a–e. Zenker's diverticula. These are usually asymptomatic in younger persons (as in **a** and **b**). In a young person Zenker's diverticulum is usually the only abnormality found. This is in contrast to in the elderly (as in **c** and **d**). In this patient there is concomitant dysfunction with pooling of contrast medium in the vallecula and in the piriform sinuses. There is also misdirected swallowing. Although the diverticulum is bigger in the young patient in (**a**), it is likely that most of the symptoms are due to concomitant pharyngeal dysmotility. However, in elderly patients the diverticulum might be huge as in (**d**). The diverticulum dislocates the cervical esophagus anteriorly and it was obvious during the examination that there was an obstruction for bolus passage

9.4 Examination Techniques

The examination technique in the elderly does not differ from that in young individuals. However, many elderly people are severely impaired and cannot cooperate. The examination should be custom-tailored to the patient's symptoms and in fact in a very impaired elderly patient there are very few relevant clinical questions that should be addressed. Therefore, such an examination is usually very easy to perform.

9.5 The Non-dysphagic Elderly

An important aspect is of course the prevalence of videofluorographic abnormalities in non-dysphagic elderly. It has been shown that only 16% of the population of aged (mean age 83) were appreciated as normal (Ekberg and Feinberg 1991). Oral and pharyngeal dysfunction was very prevalent in that study (63% and 25%, respectively) (Fig. 9.1). Combined dysfunction was present in 60%. 65% showed bolus misdirection into the airways (20% penetration into the vestibule, 45% minor aspiration to the trachea). No major aspiration was observed. Misdirection was due to oral dysfunction four times as often as pharyngeal and twice as often as combined oral and pharyngeal. In that study 36% had dissociation between the oral and pharyngeal stage. This was the pathophysiology of oral dysfunction leading to misdirected swallowing. However, in that study, half had a history of neurologic disease such as dementia, Parkinson's and stroke, although none had a history of dysphagia or swallowing impairment. Pharyngeal constrictor paresis and lingual dysfunction was much more common in those with neurologic disease. The high frequency of abnormalities that was observed in that study may have a number of explanations. Many elderly individuals do not admit to being dysphagic even when it is obvious. Caregivers are relatively poor at detecting dysphagia unless they have been specifically trained (Ekberg et al. 2002).

Altered oropharyngeal function has been documented in asymptomatic elderly volunteers (Pontoppidan and Beecher 1960; Baum and Bodner 1983; Sonies et al. 1984; Borgström and Ekberg 1988b; Tracy et al. 1989; Ekberg and Feinberg 1991; Robbins et al. 1992). Such changes may be due to normal age-related changes in tissues, muscles and neuromorphodynamics. It is not clear what is "normal decline" in oropharyngeal function among elderly and what is really the result of disease processes which definitely is more common in the elderly, particularly in the central nervous system (Levine et al. 1992; Buchholz 1992). Again, it is very important to try to compare symptoms with morphodynamic findings (Sheth and Diner 1988; Donner and Jones 1991). There is a confusing overlap of clinical, videofluoroscopic and magnetic resonance imaging (MRI) findings in elderly individuals with and without oropharyngeal dysphagia/dysfunction.

9.6 Dementia

Because dementia is the most common neurologic disease associated with misdirected swallowing it is of interest to study such a population more closely (Feinberg et al. 1992). In Feinberg's study only 7% were appreciated as normal during videofluoroscopy. Oral stage dysfunction was found in 73%, pharyngeal dysfunction in 43%, pharyngoesophageal abnormalities in 33% and combined dysfunction in 42%. When only one stage was abnormal, again oral (36%) was much more common than pharyngeal (14%). Common neuropsychiatric features of dementia can explain the oral stage dysfunction that was observed in that study. Patients frequently seem to be displaying agnosia (inability to recognise familiar stimuli or situation), dyspraxia (inability to perform coordinated movements) and abulia (psychomotor retardation). Inappropriate ingestion behaviour appears to be secondary to faulty judgement and lack of impulse control. Transitional phase dissociation was the most common oral abnormality, seen in 44% of patients. Volicer et al. (1989) have suggested that Alzheimer patients "simply have forgotten how to initiate the swallowing reflex" and a high frequency of dissociation in the above cited study may reflect such a deficit.

A recent study found correlation between the impairment of the oropharyngeal swallowing phase and the presence of unidentified objects observed incidentally on head MRI performed on elderly subjects (Levine et al. 1992). Another study found alterations of pharyngeal clearance and swallowing pressure in patients with prominent cervical osteophytic formations (Ohmae et al. 1993; Strasser et al. 2000).

9.7 The Ageing Esophagus

The term "presbyesophagus" has been used to describe esophageal motility abnormalities in elderly. However, subsequent studies showed that dysmotility does not occur secondarily to ageing as such. However, esophageal diseases are common in all age groups including the elderly. Some diseases have a relative frequency that increases with age such as adenocarcinoma. One must also take into account that an elderly patient with suspected achalasia is much more likely than a younger one to have a distal esophageal malignancy. Moreover, long-standing achalasia that occurs in an elderly patient may develop a secondary malignancy.

Symptoms of well-known diseases usually have a more complex presentation in the elderly. Therefore, chest pain due to esophageal dysmotility or gastroesophageal reflux disease may be much more difficult to distinguish from coronary artery disease. Moreover, chronic disorders present for a long time in the elderly are more likely to cause complications. This may be true for Barrett's esophagus and adenocarcinoma of the esophagus. Esophageal dysmotility is a major problem in the elderly. The symptoms are characteristically related to abnormal transportation of ingested material through the esophagus. Cardinal symptoms are chest pain and/or vomiting. The major differential diagnostic problem is to detect any underlying mechanical obstruction such as reflux (or other) stricture and malignancies. Characteristically strictures are symptomatic for solid foods but not for liquids. Dysmotility is usually equally symptomatic for liquids and solids. It is always important to consider endoscopy in this age group. If contraindicated or not available the radiologic study must include morphologic evaluation.

The effect of aging on the esophageal motor function has been studied by several authors. Non-propulsive, often repetitive contractions are numerous in the elderly (Soergel et al. 1964; Zboralske et al. 1964). Tertiary contractions and delay of the esophageal emptying, as well as dilatation of the esophagus, are also commonly seen. It has also been shown that the distal esophageal peristaltic amplitude is significantly higher in the elderly compared to younger (Richter et al. 1987). Interestingly, however, the proximal esophageal contractile amplitude did not increase with age. Others have shown that the lower esophageal sphincter resting pressure does not differ between young and elderly (Shaker 1993).

Esophageal dysmotility is one of the major reasons for drug induced esophagitis. Commonly reported drugs are NSAID, tetracycline derivates, potassium chloride and now also alendronate. Esophageal injury may be related to acidic pH in some of these drugs. However, potassium chloride causes injury by acting on smooth muscles, particularly on the small arteriole in the mucosa and submucosa. Thereby causing an ischemic lesion which may lead to fibrosis and stricture. Such drug induced esophagitis may be overcome if the patient takes precautions in terms of drinking before and after ingestion, and also to ingest in an upright position.

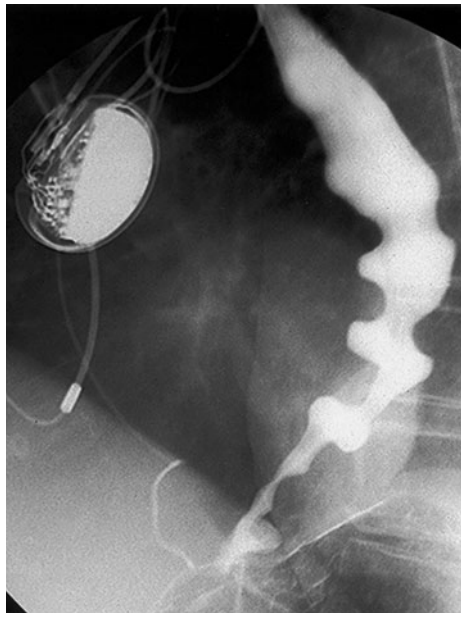


Fig. 9.4. Esophageal dysmotility is common in the elderly. This patient shows multiple non-propulsive contractions of the distal esophagus. This may, as in this patient, be very symptomatic. This 81-year-old woman vomited halfway through every meal. The barium study shows retention due to the motor dysmotility. Other patients might show similar esophageal dysfunction but do not vomit and are not otherwise symptomatic either

One common observation in the elderly is so-called corkscrew esophagus. It has a very impressive radiologic appearance. It may or may not be symptomatic. Control of motor activity in peristalsis in the esophagus is a complex interaction of excitatory and inhibitory stimuli. It should be remembered that denervation of smooth musculature leads to contraction. It may well be that these patients have lost contact between the dorsal motor nucleus of the vagus and the smooth muscle of the esophagus and that we are observing the activity of the enteric nervous system. Such corkscrew esophagus is not the same as nutcracker esophagus seen in young patients. Nutcracker esophagus has normal transportation but an increased contraction pressure. Diffuse esophageal spasm (DES)

and corkscrew contractions differ in such a way that it is considered that in the DES the peristaltic contraction obliterates the lumen, while in the corkscrew esophagus contraction does not obliterate the lumen. However, DES and curling or corkscrew dysfunction may be closely related. They both fall into the category of spastic esophageal dysfunction.

9.8 Gastroesophageal Reflux

It is important to realise that gastroesophageal reflux disease may cause strictures in the esophagus as frequently in the elderly as in younger patients. A key to the diagnosis here is of course endoscopy, but also double contrast examination. The history taking in these patients must focus on other classic symptoms or gastroesophageal reflux (Castell 1990; Fulp et al. 1990; Hey et al. 1982; Kikendall et al. 1983; McCord and Clouse 1990; Semble et al. 1989; Siebens et al. 1986; Tucker et al. 1978).

References

Baum BJ, Bodner L (1983) Aging and oral motor function: evidence for altered performance among older persons. J Dent Res 62:2–6

Borgström PS, Ekberg O (1988a) Pharyngeal dysfunction in the elderly. J Med Imag 2:74–81

Borgström PS, Ekberg O (1988b) Speed of peristalsis in pharyngeal, constrictor musculature: Correlation to age. Dysphagia 2:140–144

Buchholz D (1992) Editorial: Dysphagia 7:148–149

Castell DO (1990) Esophageal disorders in the elderly. Gastroenterol Clin North Am 19:235

Colodny N (2001) Effects of age, gender, disease, and multisystem involvement on oxygen saturation levels in dysphagic persons. Dysphagia 16:48–57

Cook IJ, Dent J, Collins SM (1989) Upper esophageal sphincter tone and reactivity to stress in patients with a history of globus sensation. Dig Dis Sci 34:672–676

Dodds WJ, Logemann JA, Stewart ET (1990) Radiologic assessment of abnormal oral and pharyngeal phases of swallowing. AJR 154:965–974

Doggett DL, Tappe KA, Mitchell MD, Chapell R, Coates V, Turkelson CM (2001) Prevention of pneumonia in elderly stroke patients by systematic diagnosis and treatment of dysphagia: an evidence-based comprehensive analysis of the literature. Dysphagia 16:279–295

Donner MW, Jones B (1991) Aging and neurological disease. In: Jones B, Donner MW (eds) Normal and abnormal swallowing: imaging in diagnosis and therapy. Springer-Verlag, Berlin Heidelberg New York

Ekberg O, Feinberg MJ (1991) Altered swallowing function in elderly pateints with dysphagia: Radiographic findings in 56 patients. AJR 156:1181–1184

Ekberg O, Hamdy S, Woisard V, Wuttge-Hannig A, Ortega P (2002) Social and mental burden of dysphagia: Its impact on diagnosis and treatment. Dysphagia 17:139–146

Feinberg MJ, Ekberg O (1991) Videofluoroscopy in elderly patients with aspiration: importance of evaluating both oral and pharyngeal stages of deglutition. AJR 156:293–296

Feinberg MJ, Ekberg O, Segall L, Tully J (1992) Deglutition in elderly patients with dementia: findings of videofluorographic evaluation and impact on staging and management. Radiology 183:811–814

Feinberg MJ, Knebl J, Tully J (1996) Prandial aspiration and pneumonia in an elderly population followed over 3 years. Dysphagia 11:104–109

Fulp SR, Dalton CB, Castell JA, Castell DO (1990) Aging-related alterations in human upper esophageal sphincter function. Am J Gastroenterol 85:1569–1572

Groher ME (1983) Mechanical disorders of swallowing. In: Groher ME, (ed) Dysphagia: diagnosis and management. Butterworth, Stoneham MA, pp 61–84

Groher ME, Bukatman R (1986) The prevalence of swallowing disorders in two teaching hospitals, Dysphagia 1:3–6

Hey H, Jorgensen F, Sorensen K, et al. (1982) Esophageal transit of six commonly used tablets and capsules. Br Med J 285:717

Hirst LJ, Ford GA, Gibson GJ, Wilson JA (2002) Swallow-induced alterations in breathing in normal older people. Dysphagia 17:152–161

Jones B, Donner MW (1991) Adaptation, compensation, and decompensation. In Jones B, Donner MW (eds) Normal and abnormal swallowing: imaging in diagnosis and therapy. Springer Verlag, New York

Kendall KA, Leonard RJ (2001) Pharyngeal constriction in elderly dysphagic patients compared with young and elderly nondysphagic controls. Dysphagia 16:272–278

Kikendall JW, Friedman AC, Oyewole MA, et al. (1983) Pill-induced esophageal injury: case reports and review of the medical literature. Dig Dis Sci 28:174

Kirkwood TBL (2000) Biological origins of ageing. In: Grimley Evans J, Franklin Williams F et al. (eds) Oxford textbook of geriatric medicine. (2nd edn. chap. 2.1) pp 35–42, chap. 2.1 Oxford University Press, Oxford

Langmore SE, Terpenning MS, Schork A, Chen YM, Murray JT, Lopatin D, Loeshe WJ (1998) Predictors of aspiration pneumonia: how important is dysphagia? Dysphagia 13:69–81

Levine R, Robbins JA, Maser A (1992) Periventricular white matter changes and oropharyngeal swallowing in normal individuals. Dysphagia 7:142–147

Logemann JA (1983) Evaluation and treatment of swallowing disorders. College Hill Press, San Diego, CA pp 64–69

Logemann JA (1990) Effects of aging on the swallowing mechanism. Otolaryngol Clin North Am 23:1045–1056

McCord GS, Clouse RE (1990) Pill-induced esophageal strictures: clinical features and risk factors for development. Am J Med 88:512

Nilsson H, Ekberg O, Bülow M, Hindfelt B (1997) Assessment of respiration during video fluoroscopy of dysphagic patients. Acad Radiol 4:503–507

Ohmae Y, Inouye T, Kitahara S (1993) Relationship between cervical osteophytes and globus sensation: a study based on altered swallowing function. Nippon Jibiinkoka Gakkai Kaiho96:379–386

Perlman AL, Guthmiller Schultz J, VanDaele DJ (1993) Effects of age, gender, bolus volume, and bolus viscosity on oropharyngeal pressure during swallowing. J Appl Physiol 75: 33–37

Pontoppidan H, Beecher HK (1960) Progressive loss of protective reflexes in the airway with advance of age. JAMA 174: 2209–2213

Richter JE, Wu WC, Johns DN, Blackwell JN, Nelson JL, Castell JA, Castell DO (1987) Esophageal manometry in 95 healthy adults volunteers. Dig Dis Sci 32:583–592

Robbins J, Hamilton JW, Lof GL, Kempster GB (1992) Oropharyngeal swallowing in normal adults of different ages: Gastroenterology 103:823–829

Schoenen J (1991) Clinical anatomy of the spinal cord. Neurol Clin 9:503–532

Semble EL, Wu WC, Castell DO (1989) Nonsteroidal antiinflammatory drugs and esophageal injury. Semin Arth Rheum 19:99

Shaker R (1993) Effect of aging on the deglutitive oral, pharyngeal and esophageal motor function. DRS, 2nd annual scientific meeting, DRS, Lake Geneva, Wisconsin, Oct 22–24, 1993

Shaker R, Cook IJS, Dodds WJ, Hogan WJ (1988) Pressureflow dynamics of the oral phase of swallowing. Dysphagia 3:79–84

Shaker R, Dodds WJ, Podursan MC, et al. (1990) Effect of aging on pharynx and upper esophageal sphincter (UES) (abstract). Gastroenterology 98:A432

Shaker R, Ren J, Podvrsan B, Dodds WJ, Hogan WJ, Kern M, Hoffmann R, Hintz J (1993) Effect of aging and bolus variables on pharyngeal and upper esophageal sphincter motor function. Am J Physiol 264:G427–G432

Shaw DW, Cook IJ, Dent J, Simula ME, Panagopoulos V, Gabb M, Shearman DJ (1990) Age influences oropharyngeal and upper esophageal sphincter function during swallowing: Gastroenterology 98:A390

Sheth N, Diner WC (1988) Swallowing problems in the elderly. Dysphagia 2:209–215

Siebens H, Trupe E, Siebens A, et al. (1986) Correlates and consequences of eating dependency in institutionalised elderly. J Am Geriatr Soc 34:192

Soergel KH, Zboralske FF, Amberg JR (1964) Presbyesophagus: Esophageal motility in nonagenarians. J Clin Invest 43:1472–1479

Sonies B, Parent L, Morrish K, et al. (1988) Durational aspects of the oropharyngeal phase in normal adults. Dysphagia 3:1–10

Sonies BC, Tone M, Shawker T (1984) Speech and swallowing in the elderly. Gerodontology 3:115–123

Strasser G, Schima W, Schober E, Pokieser P, Kaider A, Denk DM (2000) Cervical osteophytes impinging on the pharynx: importance of size and concurrent disorders for development of aspiration. AJR 174:449–453

Tracy JF, Logemann JA, Kahrilas PJ, Jacob P, Kobara M, Krugler C (1989) Preliminary observations on the effects of age on oropharyngeal deglutition. Dysphagia 4:90–94

Tucker HJ, Snape WJ, Cohen S (1978) Achalasia secondary to carcinoma: manometric and clinical features. Ann Intern Med 89:315

Vijayashankar N, Brody H (1979) A quantitativ estudy of the pigmented neurons in the neuclei locus coeruleus and subcoeruleus in man as related to aging. J Neuropathol Exp Neurol 38:490–497

Volicer L, Seltzer B, Rheaume Y, et al. (1989) Eating difficulties in patients with probable dementia of the Alzheimer type. J Geriatr Psychiatry Neurol 2:188–195

Wilson JA, Pryde A, Macintyre CCA (1989) Normal pharyngoesophageal motility: a study of 50 healthy volunteers. Dig Sis Sci 34:1590–1599

Zboralske FF, Amberg JR, Soergel KH (1964) Presbyesophagus: cineradiographic manifestations. Radiology 82:463–467

10 The Clinical and Radiological Approach to Dysphagia

Peter Pokieser and Martina Scharitzer

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10.1 Introduction

The intention of this chapter is to introduce a multidisciplinary diagnostic work-up and, in particular, to present a practical and structured radiological approach. Swallowing disorders are common. US statistics indicate that more than 5% of the population have swallowing difficulties, in hospitals up to 50% (Logemann 1995).

P. Pokieser, MD
Professor, Department of Radiology, University of Vienna, Währinger Gürtel 1820, 1090 Vienna, Austria
M. Scharitzer, MD
Department of Radiology, University of Vienna, Währinger Gürtel 1820, 1090 Vienna, Austria

The swallowing tract crosses many anatomic regions. Liquid and solid food has to be transported properly from the oral cavity into the pharynx and through the esophagus into the stomach. Thus, a wide variety of diseases may affect deglutition, resulting in a multidisciplinary work-up of dysphagic patients. Videofluoroscopy (VF) of deglutition is the method of choice to investigate the whole swallowing tract in a "one stop shopping" fashion. VF can depict pathologic findings of morphology and function as a basis to decide a further specialized work-up.

10.2 Symptoms of Swallowing Disorders

Establishing medical history is the first step in the investigation of patients with swallowing disorders to tailor the examination individually and to correlate the specific radiological findings to the patient's symptoms. A questionnaire helps to structure the patients history and should include the duration and onset of swallowing disorders, the pattern of swallowing events, the location of symptoms, the consistency of foods that lead to swallowing difficulties, as well as history of aspiration, regurgitation, coughing, pneumonia and previous operations affecting the upper gastrointestinal tract and neurological diseases.

For an adequate work-flow of diagnostic tests and therapeutic concepts the patient's history has to be differentiated into practical categories. With increasing experience with this patient group the investigator may step further into the "art and science of history taking in the patient with difficulty swallowing" (Table 10.1) (Hendrix 1993).

For the imaging specialist, the patient's history guides the design of the VF examination and has to be integrated critically into the interpretation of the study. Do the VF findings or other test results explain the patient's symptom? (Ekberg and Pokieser 1995).

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Table 10.1. Suggested protocol for the evaluation of symptoms

D	Difficulty swallowing
Y	Yesterday compared with 2 years ago?
S	Solid, liquid or both
P	Pattern of swallowing events?
H	Heartburn history?
A	Area of symptoms?
G	Gradual or sudden onset?
I	Interventions?
A	Auxiliary clues such as weight loss?

When the patient has referred her problem, questions should focus on the presence of the symptoms or syndromes discussed in more detail in the following sections.

10.2.1 Dysphagia

Eating and drinking has to be performed without pulmonary compromise and serves for personal and social pleasure, nutrition and hydration. Any subjective feeling of disturbance is called dysphagia (Buchholz 1996).

In oropharyngeal dysphagia the patient has difficulties swallowing. Isolated oral dysphagia is uncommon and based on neurogenic disorders or diminished salivary flow. Also drugs (anticholinergics, antihistaminics, antidepressants, antihypertensives, diuretics) affect salivary flow, and neuroleptic drugs may slow or disrupt the oral phase of swallowing.

Pharyngeal dysphagia is often described by the patient as a sensation of difficult passage of the bolus through the region of the suprasternal notch, most frequently caused by neuromuscular disorders causing weakness and/or incoordination of the striated muscles of swallowing (Buchholz 1987). Less frequently structural narrowings, like a neoplasm, postoperative defects, Zenker's diverticulum or a mucosal web are found.

In esophageal dysphagia the material seems to stick along the swallowing tract, the patients may localize the site anywhere from the suprasternal notch to the epigastrium. Usually the patients cannot differentiate between a proximal or distal site of an esophageal lesion (Edwards 1974). For example, a Schatzki ring at the level of the esophagogastric junction or achalasia often produce symptoms above the suprasternal notch. Intermittent esophageal dysphagia for solid food is typical for lower esophageal rings or strictures with a remaining lumen of less than 2 cm. Rapid progress of solid food dysphagia within 3 months is often found in esophageal carcinoma. If there is no sign or proof of aspiration, esophageal dysphagia for fluids only indicates esophageal motor disorders. Depending on the severity of the motility disturbance, the latter can produce solid food dysphagia as well. Regurgitation of previously ingested food can arise during a meal from any cause or location, late regurgitation of undigested food is typical for a Zenker's diverticulum or achalasia. Complaints of sour and/or bitter material with heartburn is pathognomonic for gastroesophageal reflux (GER). Furthermore, gastroesophageal reflux disease (GERD) is the most common cause for "non cardiac chest pain". After exclusion of a cardiac cause, the esophagus has to be evaluated. A 3-week therapy with proton pump inhibitors can be an effective diagnostic and therapeutic approach. Odynophagia means painful swallowing, the pain is described as "sharp", usually indicating ulcerative mucosal lesions of the pharynx or esophagus, whereas a dull or squeezing pain is associated with esophageal spasm.

10.2.2.1 Globus Sensation

Globus is a common problem, representing about 5% of general otolaryngologic patients. A "lump in the throat", the sensation of a foreign body, sore throat, frequent throat clearing and fullness are typical complaints of these patients. Symptoms tend to occur intermittently. Often the symptom improves during eating, while a combination with dysphagia is frequently found. In 75% of 150 patients with globus as the only symptom VF could depict pathological functional and/or morphologic findings, evidence of an esophageal motor disorder was present in 47% (Schober et al. 1995). A high incidence of esophageal motility disorders in this setting was detected by manometry in 87% (Moser et al. 1991). Globus sensation seems to be a symptom of laryngopharyngeal irritation, not specific to GERD, but in which GERD plays a role (Woo et al. 1996). There remains an open discussion about the pathogenesis of globus. However, the term "globus hystericus" should be avoided and pharyngeal and/or esophageal pathologies should be ruled out according to the specific history. If patients with chest pain do not show any evidence of cardiac disease, the term "non-cardiac chest pain" is often diagnosed.

10.2.3 Aspiration

Aspiration is defined as the entry of liquid or food into the airways below the level of the glottis. Choking and/or coughing immediately following a swallow, as well as recurrent pneumonia, is suspect of aspiration. Silent aspiration may occur if the cough reflex is absent or diminished. Persons who aspirate are at increased risk for the occurrence of serious respiratory sequelae, including airway obstruction and aspiration pneumonia. The quantity, the depth of aspiration (trachea or distal airways) and the physical properties of the aspirate influence the effects of aspiration (Palmer et al. 2000). Aspiration can happen anterograde during or immediately after swallowing or as retrograde aspiration of gastric or esophageal contents. The radiologist must be aware of patients at risk of aspiration. The tailored VF study avoids severe aspiration during the examination (Jones and Donner 1988).

10.3 Multidisciplinary Evaluation of Swallowing Disorders

The radiologist should be familiar with the specific techniques of different medical fields when investigating patients with swallowing problems. There is a considerable overlap in using different clinical tests by different clinical fields, which varies from country to country. We try to clarify more the clinical interaction than the borders between clinical specialties.

General patient status includes mental and social function, physical mobility, as well as his/her nutritional and hydration status.

Examination of the chest can reveal problems with the respiratory function related to aspiration or conditions in which aspiration might cause severe problems. The cardiovascular system has to be assessed for possible sources of emboli to the brain, impairment of the musculoskeletal system can affect normal mobility, and the swallowing mechanism can be affected by different systemic diseases, such as scleroderma or muscular diseases.

The status of otolaryngologists and speech/language pathologists includes a full head and neck examination. The neck should be evaluated for masses, especially for adenopathies, enlarged thyroid and scars which indicate surgery on structures involved in swallowing. An inspection should be performed of the oral cavity, cranial nerve function, palate, pharynx and larynx with indirect laryngoscopy or by fiberoptics to assess for tumours, mucosal integrity, vocal cord motion, pooling of secretions into the vallecula or the piriform sinus, as well as sensation and voice (Sonies 1987). Stridor is a sign of upper airway obstruction and may be audible only on auscultation over the trachea, the sounds of swallowing motility and the palpation of the elevation of the hyoid and larynx are part of the dynamic clinical investigation. Fiberoptic endoscopic evaluation of swallowing (FEES) is a well established diagnostic test and enables assessment also of nasal, velopharyngeal, and laryngeal pathology of morphologic and functional changes. The flexible, light weighted instruments are inserted through the nose. Video documentation can be integrated. FEES is complementary to videofluoroscopy and the application and interpretation of results is an interdisciplinary task of otolaryngologists, speech/language pathologists and radiologists. FEES does not show the entire motion of essential foodway structures and the bolus during swallowing, but can identify aspiration and pharyngeal retentions. Firstly, results of FEES and VF are "diagnostic studies". Secondly, they allow the clinician to design an appropriate diet and compensatory manoeuvres designed to improve pharyngeal clearance and reduce aspiration. This use of VF and FEES is called "therapeutic studies", including investigations to test the effectiveness of therapy.

FEES is also used by neurologists. However, the neurologic examination is a crucial part of the multidisciplinary approach to dysphagic patients. Cerebrovascular disease, Parkinson's disease, multiple sclerosis, amyotrophic lateral sclerosis, poliomyelitis, myasthenia gravis and dementia are examples of the wide spectrum of neurological disorders which may involve deglutition.

Invasive treatment of the upper esophageal sphincter (UES) is performed by otolaryngologists, as well as general surgeons, and frequently used as therapy of Zenker's diverticulum. Different surgical strategies are under continuous discussion. Myotomy of the UES with or without resection of the diverticulum itself, myotomy with laser or with an endoscopic approach are applied to restore an adequate opening of the pharyngoesophageal segment.

Gastroenterologists and surgeons are specialized in the diagnosis and treatment of esophageal disease. Endoscopy reveals even subtle mucosal details and can take biopsies for pathologic diagnosis. But endoscopy may overlook subtle rings or stenoses,

which can be passed by the endoscope, but will hinder larger boluses of solid food. Furthermore, endoscopy cannot display the topographic relation of stenoses to the important anatomic landmarks in all cases and often cannot be forwarded distal to a narrow stenosis. VF is helpful in such instances, providing excellent topographic overviews, testing for subtle stenoses with solid bolus and examining the esophagus distal to stenoses. Benign and malignant macromorphological changes of the esophageal tube are detected by endoscopy and VF (Scharitzer et al. 2002). In esophageal motility disorders VF can detect a delayed transport for liquids and solid food as well, complementary to manometry, the gold standard to diagnose esophageal motility disorders. Manometry can be combined with VF as videomanometry. This method synchronizes the videofluoroscopic record of the bolus transport and the measurements of pressures. Evaluation of esophageal transport and gastric emptying is obtained by scintigraphy. Delayed gastric emptying, well known in diabetic disease, may interfere with esophageal transport and contribute to symptoms like dyspepsia, epigastric fullness or heartburn. pH probe studies can detect pathologic gastroesophageal reflux, while VF can describe the dynamic appearance of the esophagogastric junction during and after passage of a bolus. Hiatus hernia, cardiac insufficiency, the esophagogastric junction after operations like fundoplication, myotomy, dilatation, gastric banding and other operations are studied videofluoroscopically to rule out early and late postoperative complications, such as stenosis, leakage or perforation. VF is the diagnostic test of choice to obtain a general overview of the whole swallowing tract, to detect macropathologic changes and disordered function as well. Pertinent to the clinical problem, a tailored VF examination can be designed to be the basis for other diagnostic tests or to complete their results in a complementary way.

10.4 Imaging of Swallowing Disorders

10.4.1 Technical Considerations

Videofluoroscopy is performed with a fluoroscopy unit connected to a video recorder. The dynamic examination studies motility of the oral, pharyngeal and esophageal phases, whereas the spot film examinations demonstrate morphology. Any fluoroscopic unit that offers remote control equipment is appropriate. By using the jog-wheel function the videofluoroscopic study can be analysed frame-by-frame several times. Computer based dynamic recording on hard disk, DVD or CD is still under development and offers have to be evaluated carefully in case of interest. Continuously recording for 20 seconds seems to be the minimum, while cheap and easy storage of the examinations is mandatory.

The introduction of digital fluoroscopy and computer-based workstations offers a higher spatial resolution and new possibilities for the interpretation by lower radiation exposure at the same time. Digital images obtained during a dynamic study can be post processed and transmitted more easily.

10.4.2 Examination Technique

The approach as described by Ekberg and Pokieser (1997) is based on the patient's history, planning the investigation in detail. The radiographic examination has to include all structures involved in swallowing, from the lips to the stomach. Nevertheless, it is important to focus the examination on specific areas. In any patient with a high suspicion of laryngeal or pharyngeal disease, the laryngeal vestibule should be included in the image from the beginning. It is very common that the first swallow is the worst swallow and that only the first swallow will reveal dysfunction.

It is certainly very important to realize that there are two basically different examinations of swallow. One is customized for the diagnosis, i.e., the search for why the patient has a specific symptom (diagnostic study). This test is basically concerned with finding that particular patient's worst swallow and therefore might include manoeuvres for decompensation of a compensated swallow (Buchholz et al. 1985). This is in contrast to the diagnostic examination which is done when the dysfunction in a specific patient has been revealed. This test basically tries to reveal the patient's best swallow and therefore always includes manoeuvres for compensation of a decompensated swallow (therapeutic study). It is always important to observe as many swallows as possible, as dysfunction may be intermittent. Moreover, the benefits of therapeutic manoeuvres are notoriously difficult to assess. The performance of dynamic studies turns the investigator from a photographer into both a film director and camera operator.

No special preparation is needed for the radiological examination of the upper gastrointestinal tract.

The patient is examined with his dentures or other oral appliances in place so that the patient's swallow is as normal as possible. Nasogastric feeding tubes should be removed prior to the investigation, but due to the discomfort during reinsertion of tubes, this has to be decided with regard to the clinical problem. The patient should stand or sit on the footboard of the fluoroscopic table. Esophageal motility can be depicted best in the horizontal position, when the contrast material is pushed mainly by esophageal motility (Fig. 10.1).

Debilitated patients can be examined on a specially designed chair. Patients should not experience stress during swallowing, and the investigator has to care to provide a quiet and comfortable environment during the study.

10.4.2.1 The "Seven Functional Units"

The systematic analysis procedure known from X-ray reporting proved to be useful for reporting film sequences as well.

The radiological substrate of this analysis is the movements of anatomical structures that cause the formation and continuous propulsion of the bolus of contrast medium. Physiologically, the act of swallowing is divided into three phases: the oral, the pharyngeal and the esophageal phase. The duration of the oral phase may be determined arbitrarily. The pharyngeal phase takes less than 1 s. The esophageal phase takes approximately 10 s.

Fig. 10.1. Prone oblique position: Contrast material is given by a straw. Abnormal structural movements may be diagnosed. Further misdirection or retention of a bolus can be documented. The seven functional units may serve as a basis for the structured radiological report on deglutition

The esophageal phase is about ten times as long as the pharyngeal phase.

For radiographic analysis of the act of swallowing, it is useful to summarise the involved anatomical structures into seven functional units (Pokieser et al. 1995).

The description of normal and pathological functional findings of the seven functional units of the act of deglutition is a simplification of the subject, but does include the most important clinical findings needed by those embarking on routine diagnosis of the act of swallowing (Fig. 10.1).

10.4.2.2 Design of Videofluoroscopic Scripts

A standard examination of these seven functional units is performed by lining up various film sequences of the video recording of the act of swallowing and fluoroscopy. During the pharyngeal phase of swallowing, the functional units should be filmed in a stationary position, as the recording will be blurred and rendered unusable for study if the central beam is moved. During the esophageal phase, the central beam may follow the bolus, because the esophageal propulsion is relatively slow (~ 4 cm/s).

Swallowing Disorders Without Suspicion of Aspiration

Patients suffering from dysphagia, globus, chest pain or other clinical conditions related to swallowing may have no clinical symptoms of aspiration. These patients should swallow boluses of normal size and should be investigated in all standard positions as mentioned below. The examination starts with films of the erect standard positions, followed by tests of esophageal motility in the horizontal, supine and prone positions. Plain films in double contrast technique should be added, according to the clinical question. Effervescent powder disturbs the standardized examination of the esophageal tube and should be given after studying the esophageal motility in the horizontal position. The passage of contrast material should be followed to the duodenojejunal junction.

Swallowing Disorders with Suspicion of Aspiration

The investigation is restricted to erect standard projections, if aspiration occurs. With increasing amounts of contrast material, severe aspiration can be avoided by stopping any increase when the patient aspirates. Different consistencies are applied in diagnostic studies pertinent to the symptom, and in therapeutic studies to

find out the best consistency without aspirating. This is best done in collaboration with a speech and language pathologist, who has complementary clinical information and interest in the therapeutic approach.

From a systematic point of view, every film scene may be defined by three characteristics: First, the selected section for imaging and the patients *standard position*; second, the selected *type of contrast medium*, and finally third, the *amount* of contrast medium.

10.4.2.3 Standard Positions for Videofluoroscopy (Fig. 10.2–10.7)

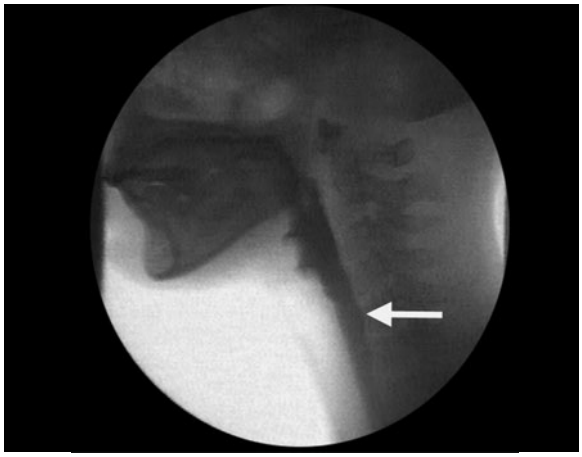


Fig. 10.2. First standard position: Overview of the lateral oral cavity and pharynx. In this position, the pharynx is shown in the largest possible section of the image in a way that the oral cavity and, in the caudal aspect, also the upper esophageal sphincter is included (*arrow*). The patient is examined in upright position, either standing or sitting. Usually the patient is turned to the right side, slightly oblique. It is useful to repeat this scene after turning the patient left, if stenoses need to be ruled out

Fig. 10.3. Second standard position: This setting shows the upper esophageal sphincter in the lateral view, slightly oblique (*arrow*). The upper esophageal sphincter can be evaluated especially well in this targeted image of the cervicothoracic junction

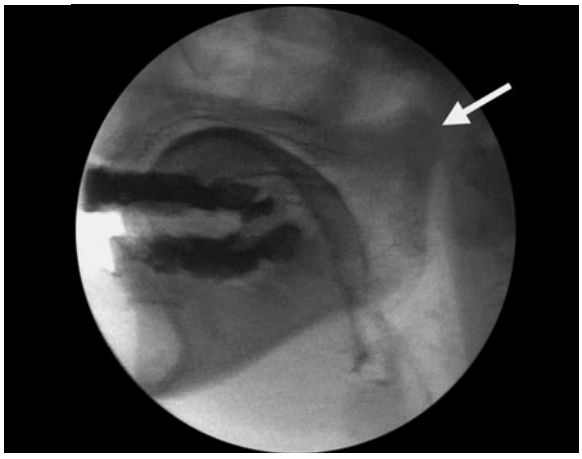


Fig. 10.4. Third standard position: Sometimes it is useful to view the oral cavity separately. In doing so, the movement of the soft palate during speech can be visualized. However, the pharyngeal phase is largely eliminated in this setting. While speaking, the soft palate rises up to the posterior wall of the nasopharynx, while the patient says "Kathy" (*arrow*)

Fig. 10.5. Fourth standard position: Frontal view of the oral cavity, the pharynx and the cervical esophagus. The symmetry of the passage has to be documented

Fig. 10.6. Fifth standard position. This image shows the middle and lower esophagus in the erect left posterior oblique position. It is useful to repeat this scene in the right posterior oblique position to obtain two different projections of the esophageal phase. Gravity enforces the passage of contrast material in the erect position. With a single act of swallowing, the passage through the lower esophageal sphincter is shown (*arrows*). The patient is asked to swallow just once, to hinder repeated superimpositions of peristaltic waves. Practical advice: about 3 s after the pharyngeal phase in the first or second standard position you develop the fifth standard position. by following the bolus. This can be done when no abnormal findings were visible during the pharyngeal phase; then stop the movement of the central beam, when the esophagogastric junction is visible

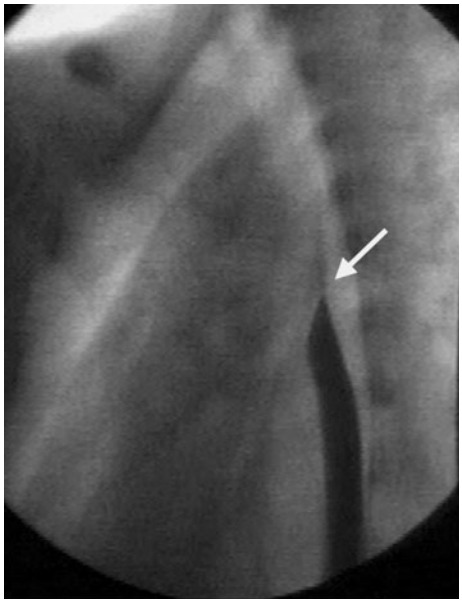


Fig. 10.7. Sixth standard position: The esophageal passage in prone position allows the peristaltic wave of the esophagus to be visualised. Repeated in the supine position, the esophageal transport is visualised in a different way. Hernias, rings and other findings are often seen in one position only - prone or supine. The cranially V-shaped peristaltic wave (*arrow*) is followed downwards from the top. As advised for the fifth standard position, you may follow the peristaltic wave with the central beam. The central beam should not be moved, during visualisation of the dynamic movements of the esophagogastric junction

10.4.2.4 Type of Contrast Medium

We use high-density barium suspension to visualize the morphology and function from the oral cavity to the esophagus.

Non-ionic iodinated low-osmolar or iso-osmolar water-soluble contrast material is necessary for patients with clinical suspicion of aspiration or perforation. Hyperosmolar iodinated contrast medium is contraindicated in patients with suspicion of aspiration. In addition, solids are indicated to show a stricture, a solid-induced spasm or dysphagia, as well as for postoperative control studies. Therefore, a piece of bread with barium or placebo tablets with a 13-mm diameter can help in the evaluation of solid-induced abnormalities. The use of different consistencies is extremely helpful in patients with aspiration of only liquid boluses in order to assess the further therapeutic and dietetic management.

10.4.2.5 Amount of Contrast Medium

The normal amount of a single swallow of an adult patient is about 15 ml. However, a healthy adult can manage liquid boluses up to 50 ml and more. The amount of contrast material should be varied according to the individual capabilities. The examination includes up to ten swallows of 15–30 ml of liquid barium sulfate. In the beginning, the amount of contrast media is 15 ml. In cases with suspected aspiration it is lessened to 3 ml for the first swallow. If this bolus volume is tolerated, the bolus size should be increased to 5 ml and up to 15 ml.

The contrast medium can be applied in many different ways, according to the abilities of the patient. Cups, drinking bottles, spoons or straws may suite. The best way to administer contrast material orally can be designed by the feeding history and the clinical presentation of the patient.

10.4.3 Reporting on the Seven Functional Units of Swallowing

The basic principles of the functional physiology and pathology of swallowing constitute the actual radiological basis for reporting videofluoroscopy.

The seven functional units are a radiological approach based on the dynamic radiological information (Fig. 10.8).

Often there are several functional disorders of the same functional unit in a single patient. Countless combinations of findings occur, but typical "patterns of findings" are to be observed. Step by step the seven units may be analysed for malfunction. The movements of the visible anatomical structures are the basis of the analysis, the passage of the bolus displays, whether the function of the swallowing tract is adequate for the given amount and material. Each unit represents an area, where the investigator can focus the analysis easily. Videofluoroscopy shows just a part of the complex structures below the barium coating. The strength of the method is the ability to see the entire swallowing action simultaneously with its effect on bolus transport.

10.4.3.1 Tongue – Oral Cavity

Normal Function of the Tongue (Fig. 10.9)

Incompetent Bolus Manipulation. An incompetence to manipulate the bolus summarizes many different dynamic findings (Fig. 10.10). The inability of the tongue to hold the bolus on its upper surface, fragmentation of the bolus, uncoordinated movements like tremor or undulations may be visible. When lip closure is insufficient and material runs out over the lips, this is called "drooling".

Weakness of the Tongue. Weakness of the tongue is often combined with pharyngeal weakness, retention in the valleculae can be caused by both, and overlaps cannot be differentiated (Fig. 10.11).

When weakness of the tongue is present, a high consistency of the contrast material proves to be more sensitive, like barium paste or bread with barium. Water soluble contrast material can be propelled more easily, thus masking a weakness of the tongue.

Incompetent Tongue–Palate Seal. Incompetence of the apposition of the soft palate and the tongue leads to leakage of the bolus into the oropharynx (Fig. 10.12). Weakness or postoperative defects of the tongue, the soft palate or both can cause this functional deficit. The differentiation between leakage and late triggering of the involuntary swallow can be difficult, when the oral transit time is short. This might be the case when the patient reclines the head to compensate difficulties of the oral transport.

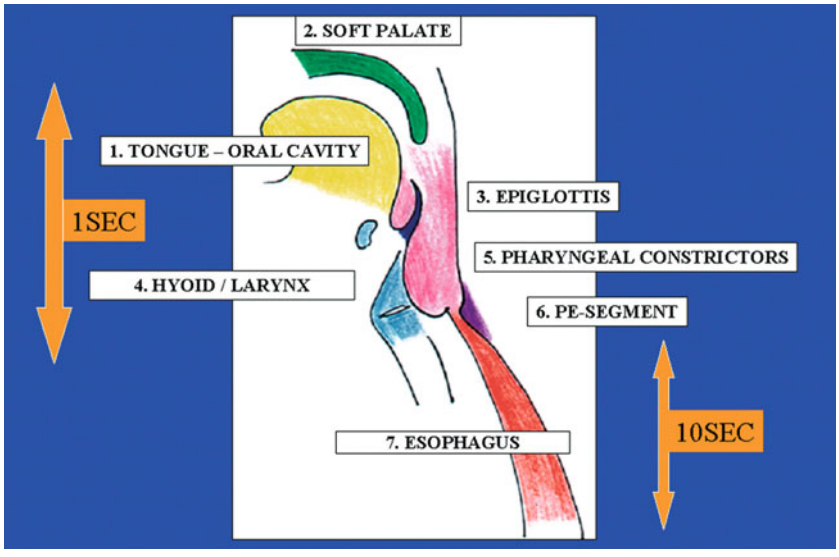
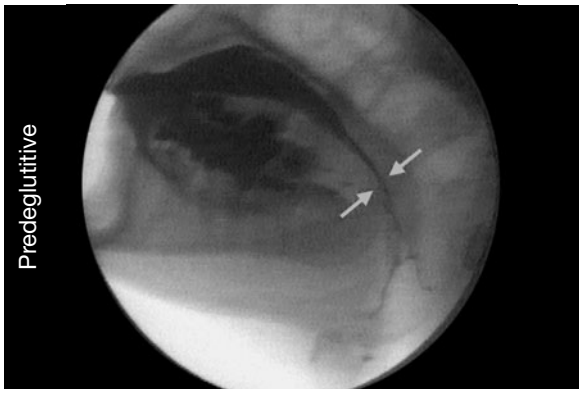


Fig. 10.8. This graph demonstrates the seven functional units of swallowing. Within 1 s six of the units complete the pharyngeal stage of swallowing – then the esophagus bridges the long distance through the mediastinum to the stomach. A complete barium swallow of about 15 ml should reach the stomach within 10 s



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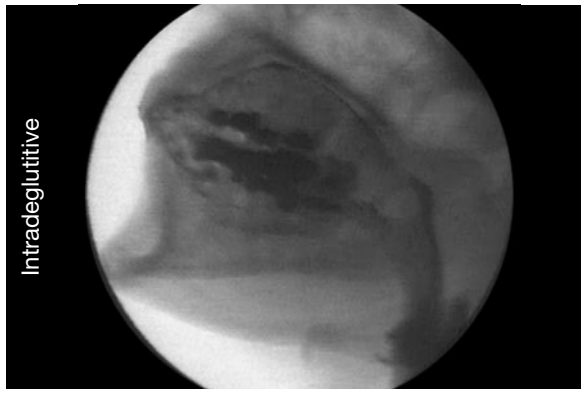
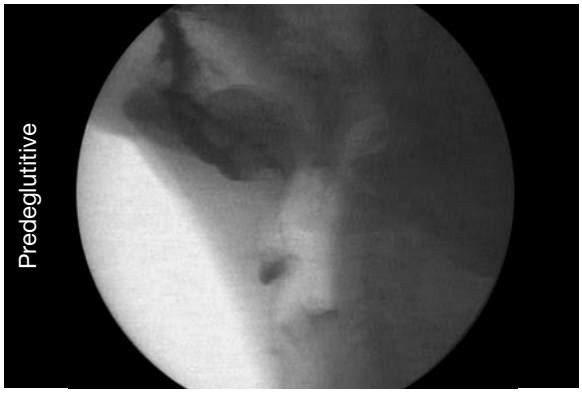


b

c

Fig. 10.9a–c. a The oral phase is started by the intake of a beverage or the ingestion of food into the oral cavity. The lips close and seal the oral cavity anteriorly. To form the bolus, the substance is first loaded on the dorsum of the tongue. This process is under voluntary control. In this process, the tongue and soft palate together seal off the posterior part of the oral cavity (*arrows*). **b** When the involuntary act of deglutition is started, the tongue performs a wave-like movement and presses the manipulated bolus along the hard palate backwards into the pharynx. **c** Only the barium coating remains in the oral cavity



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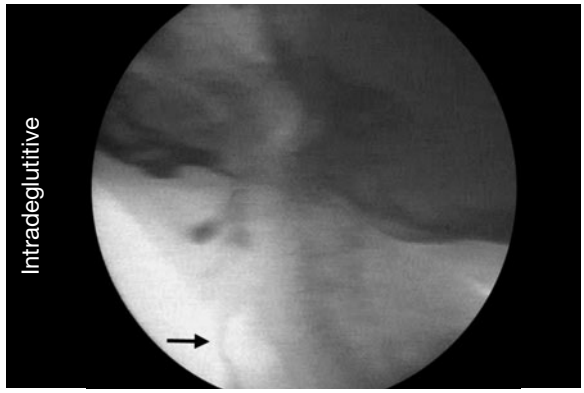

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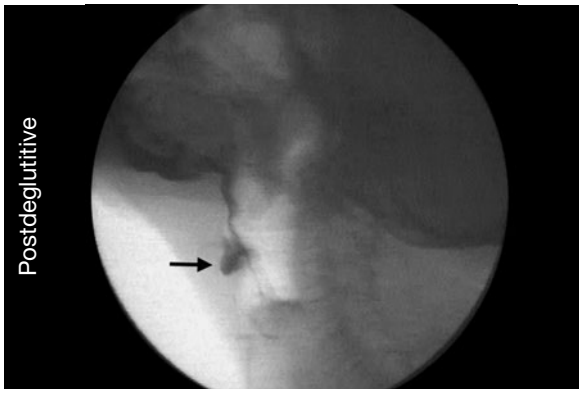
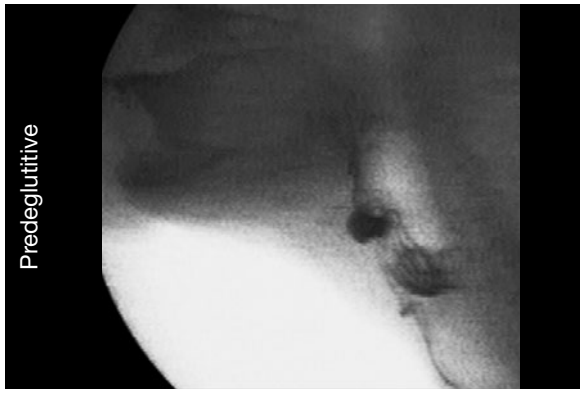


c

Fig. 10.10a–c. a This 75-year-old woman with cerebral ischaemia cannot control the bolus in the oral cavity. **b** The oral transit is achieved by inclining the head backwards. This causes some blurring and the spatial resolution is reduced. Only minimal amounts of material are transported. Aspiration occurs (*arrow*). **c** Contrast material has pooled in the lower oral cavity beneath the anterior parts of the tongue. Minor aspiration after the swallow (postdeglutitive) has occurred, and there is pooling in the valleculae (*arrow*)



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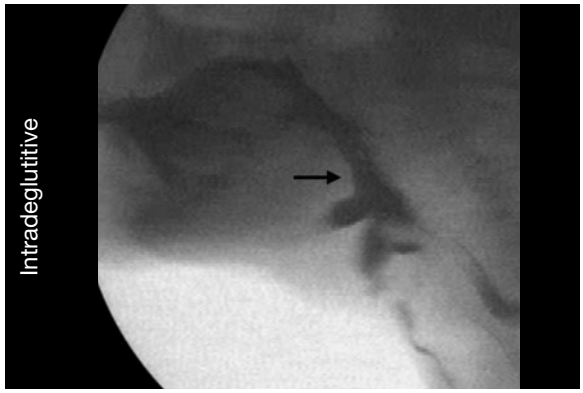

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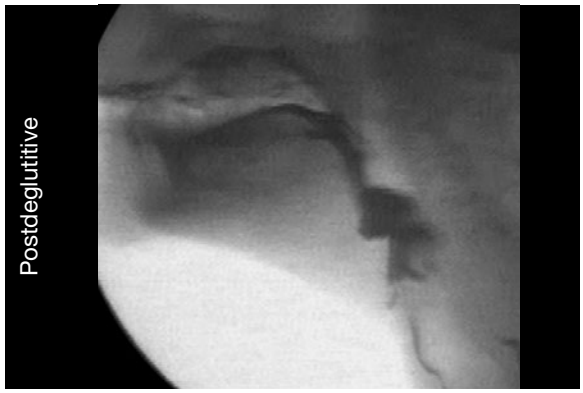


c

Fig. 10.11a–c. a An 81-year-old lady suffers from Parkinson's disease. She has already aspirated material from previous swallows. Pooling in the valleculae is present. **b** The tongue fails to reach the hard palate and cannot propel the whole bolus into the oropharynx. Dorsal excursion of the tongue appears to be limited (*arrow*). **c** Barium mixed with mucus remains stuck to the hard palate and the tongue. Retentions in the valleculae have increased

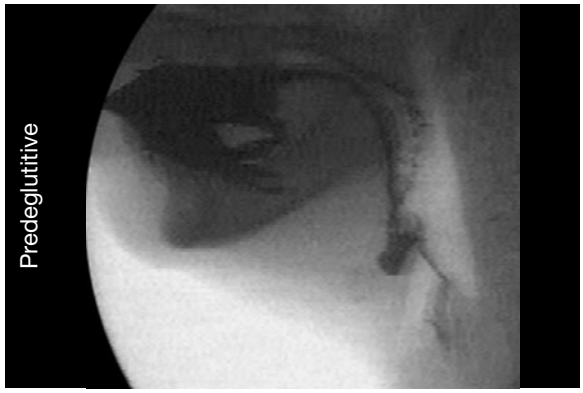
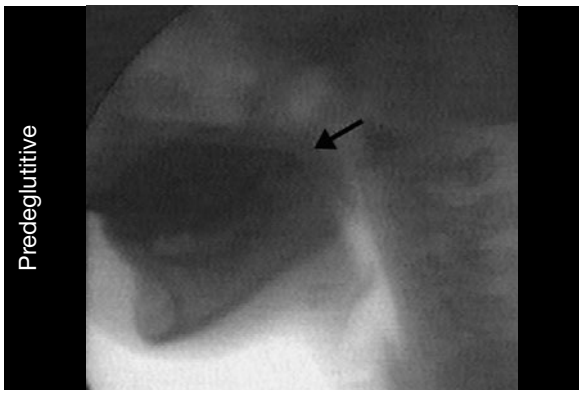


Fig. 10.12. In this 69-year old patient with cerebral ischaemia, the dorsal closure of the oral cavity is disturbed and contrast medium prematurely passes into the pharynx – so-called "leaking"

10.4.3.2 Soft Palate

Normal Function of the Soft Palate (Fig. 10.13)



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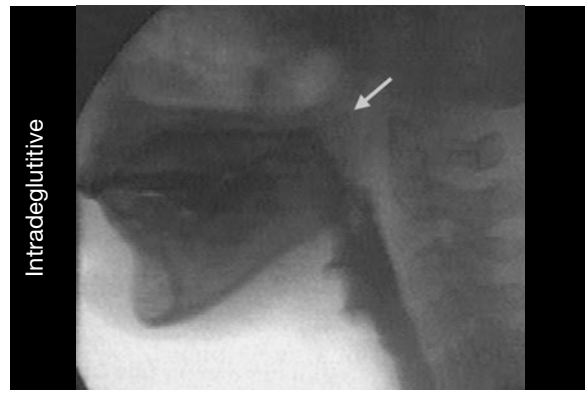

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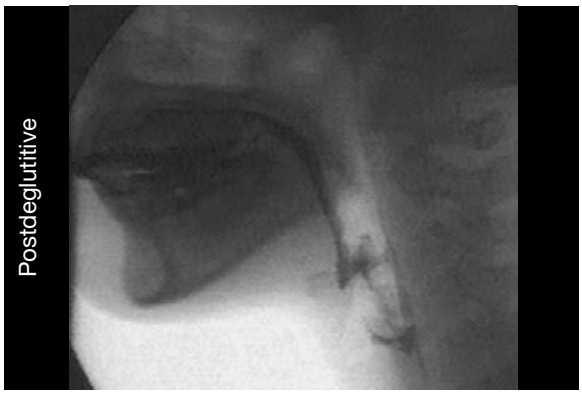


c

Fig. 10.13a–c. a Before the act of swallowing, the soft palate, in conjunction with the tongue, seals off the oral cavity dorsally (*arrow*). **b** During the pharyngeal phase the soft palate is raised to a right angle and tightens the oropharynx together with the posterior wall of the pharynx (*arrow*). The latter is called Passavant's cushion at this level, where the pharyngeal wall converges to the soft palate. Thus, food particles are prevented from entering the nasopharynx. One can test the elevation of the soft palate towards the posterior wall of the pharynx by having the patient utter words with a "k", for instance, "Kathy". **c** After the act of swallowing, the soft palate returns to its original position

a) Incomplete Elevation (Fig. 10.14)

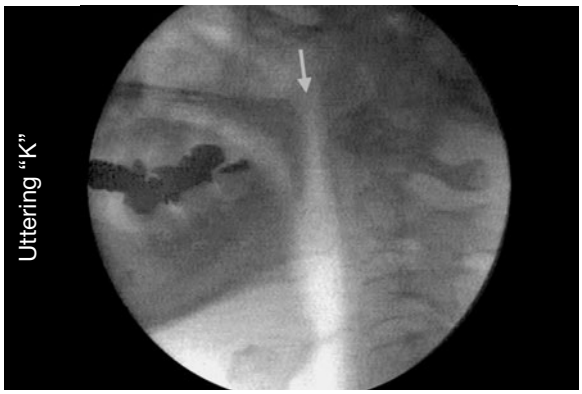


Fig. 10.14. In this 55-year-old patient with myasthenia, elevation of the soft palate is disturbed. A gap of air remains between the posterior wall of the pharynx and the soft palate (*arrow*) when the patient utters "k"

b) Insufficient Velopharyngeal Closure (Fig. 10.15)

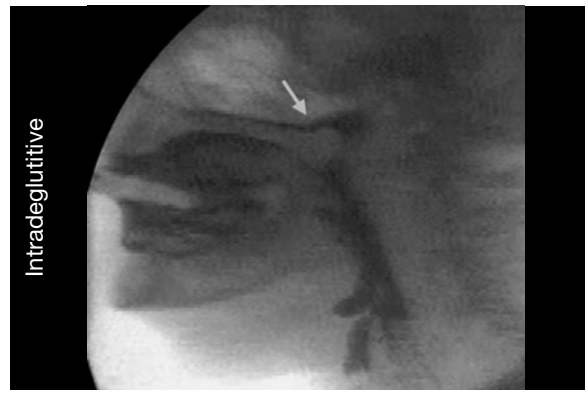


Fig. 10.15. This 53-year-old woman who had undergone surgery for a carcinoma of the right tonsil has a defect in the dorsal soft palate, which causes regurgitation. During swallowing spillage of contrast material into the nasopharynx can be observed. Contrast material has penetrated into the larynx (*arrow*).The soft palate is shortened and deformed postoperatively. Aspiration has occurred

10.4.3.3 Epiglottis

Normal Function of the Epiglottis (Fig. 10.16)

Incomplete Epiglottic Tilt. An incomplete epiglottic tilt is almost always combined with retentions in the valleculae, and often with hypopharyngeal retentions and numerous other disturbances of the pharyngeal phase (Fig. 10.17). Tumours, swelling after radiation or in inflammatory diseases such as epiglottitis, and postoperative structural deficits up to total resection should be considered.

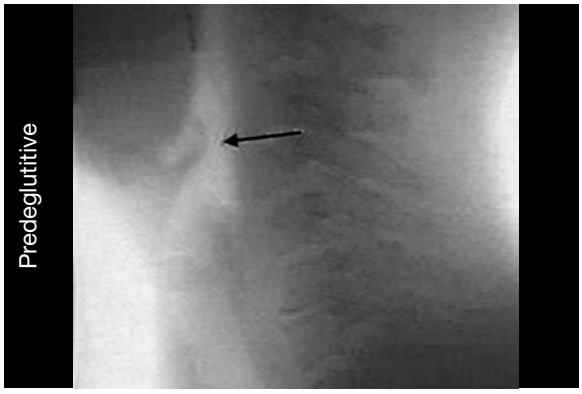
10.4.3.4 Hyoid and Larynx

Normal Function of the Hyoid and the Larynx (Fig. 10.18)

Poor Elevation. Tracheostomy, pharyngeal or laryngeal resections, radiation therapy or muscular weakness are common causes of poor movements of the epiglottis and the larynx (Fig. 10.19). Over time, this condition contributes to pharyngeal retentions and aspiration.

The next four functional disorders are related to insufficient protection of the respiratory tract, namely penetration of contrast medium into the larynx and the three basic forms of aspiration into the trachea: pre-deglutitive, intra-deglutitive and post-deglutitive aspiration.

Penetration. After penetration, we will look at the basic forms of aspiration. The terms pre-, intra- and post-deglutitive refer to the involuntary act of swallowing and the rapid, automatic motion of the larynx, hyoid and pharynx. Thus, aspiration may occur before, during or after the rapid swallowing movement (Fig. 10.20).



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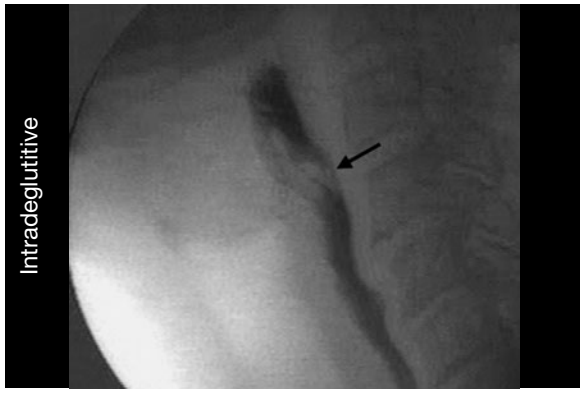

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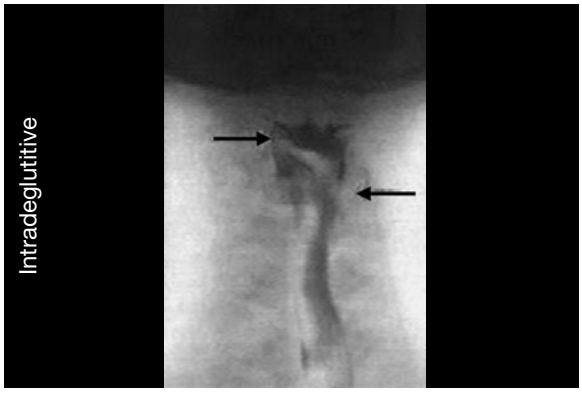
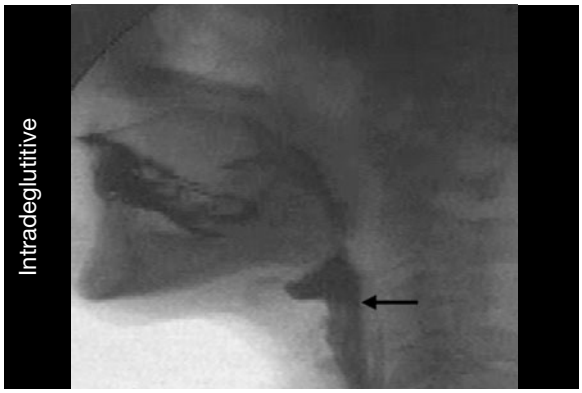


c

Fig. 10.16a–c. a The epiglottis is usually seen even without contrast medium, in the lateral view of the pharynx (*arrow*). **b** The epiglottis should tilt below the horizontal plane. The complete tilt can be seen as a small longitudinal structure at the end of the bolus passage (*arrow*).**c** Even healthy individuals with a good swallowing function occasionally reveal asymmetrical tilting of the epiglottis. In frontal views, one side tilts more deeply than the other (*arrows*). Unilateral muscular weakness can cause the same finding, but is often combined with abnormal radiological findings or other clinical signs of swallowing impairment



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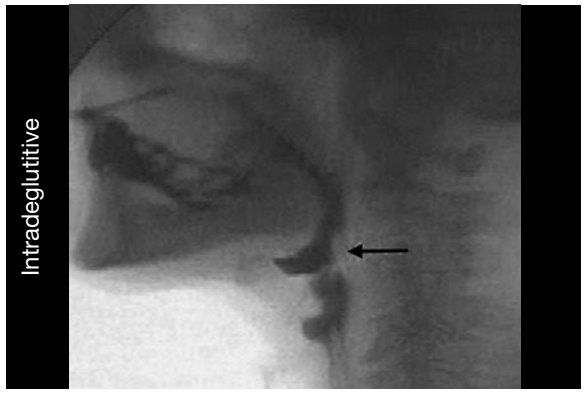

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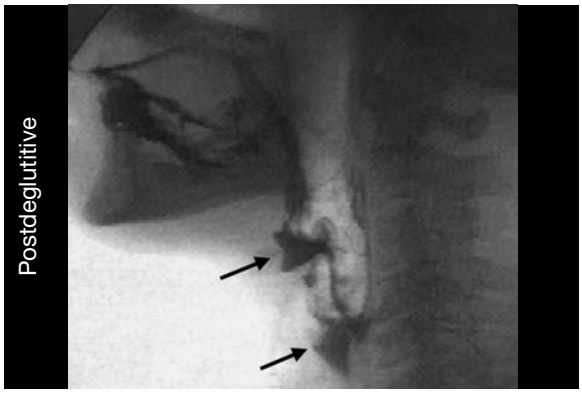
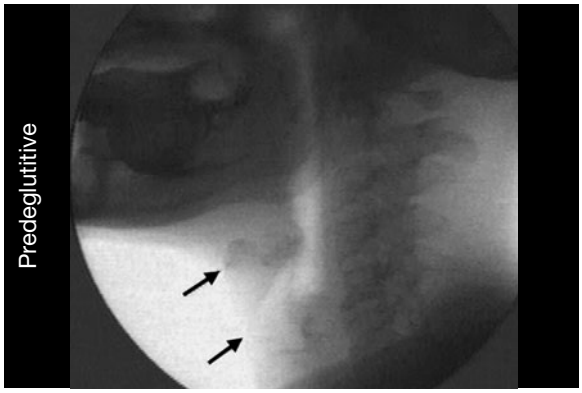


c

Fig. 10.17a–c. a, b Incomplete epiglottic tilt (*arrow*). **c** This is combined with retention in the valleculae and piriform sinuses (*arrows*)



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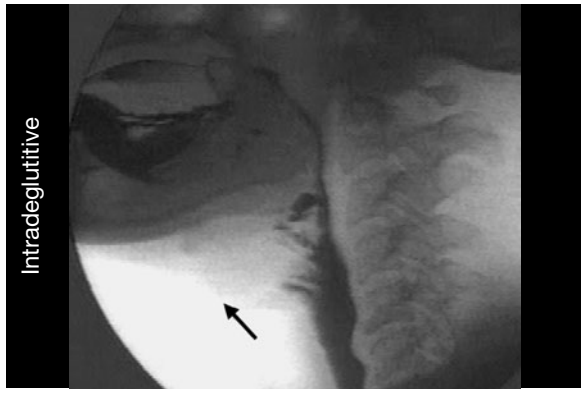

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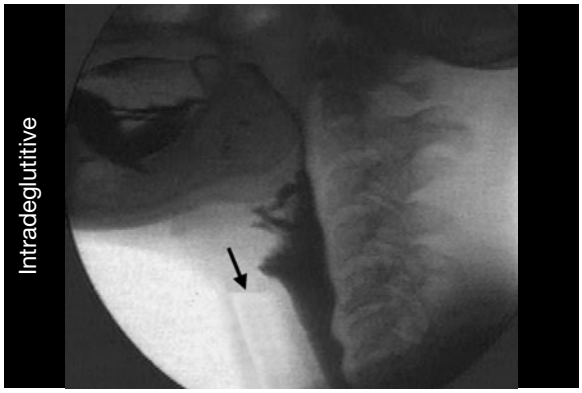
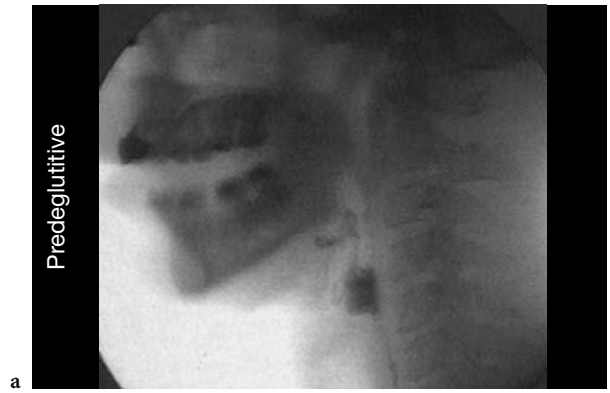


c

Fig. 10.18a–c. a The hyoid bone and the air column in the lateral projection. The hyoid is visible as a bony structure, while the larynx is represented by an air column (*arrows*). **b** To protect the respiratory tract, the hyoid and larynx move cranially and ventrally at the beginning of the involuntary act of swallowing (*arrow*). The elevation of the larynx is visible fluoroscopically. It has to be emphasised that the pharyngeal muscles are elevating simultaneously, almost invisible for videofluoroscopy. **c** The opening of the larynx is tightly closed during the movement of the larynx, the air column of the trachea is visible up to the horizontal end at the level of the vocal cord (*arrow*). During elevation, the hyoid and larynx come closer to each other. The upward movement of the larynx can be measured as an approximation. It varies widely individually and with the volume of the bolus. If the elevation is less than 1 cm it can be described as abnormal. The height of a cervical vertebral body represents another approximation to estimate the minimum of laryngeal elevation



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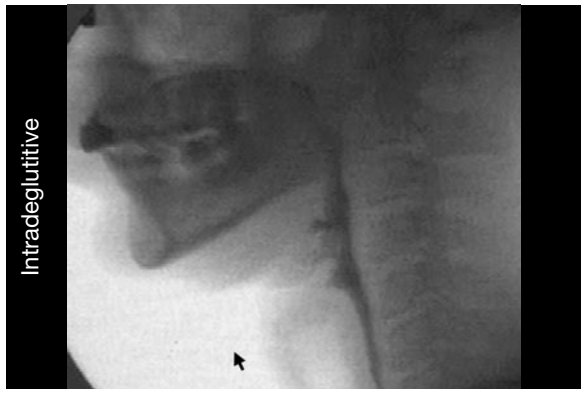

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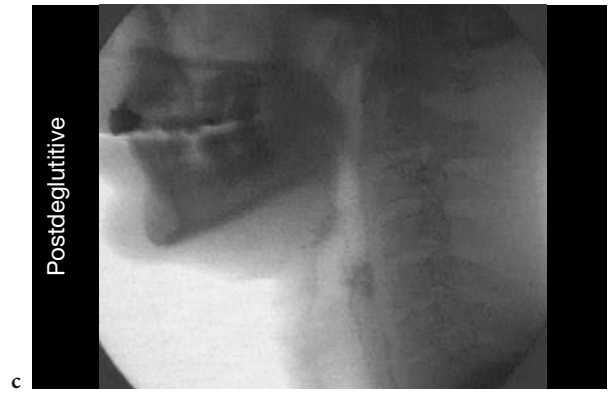
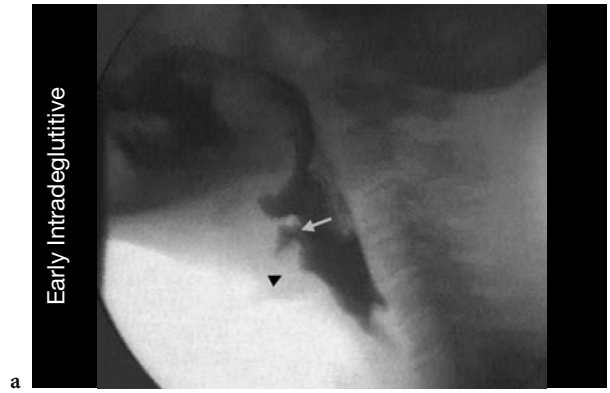


c

Fig. 10.19a–c. a In this 45-year-old man who underwent resection of a tumour of the pharynx, retentions of contrast material in the hypopharynx from previous swallows are present. **b** The larynx rises very slightly, less than 1 cm and not even 50% of the height of a cervical vertebral body (*arrow*). **c** After the swallow, some drops of contrast material have entered the larynx



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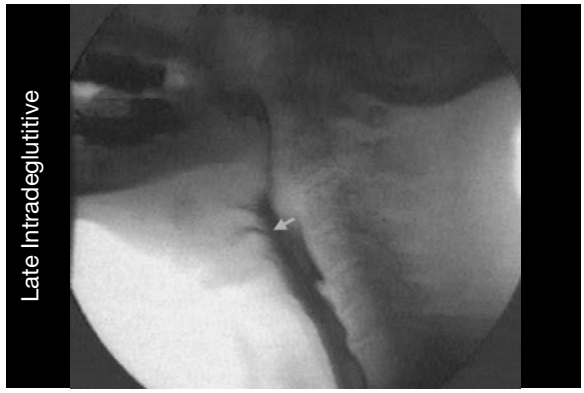

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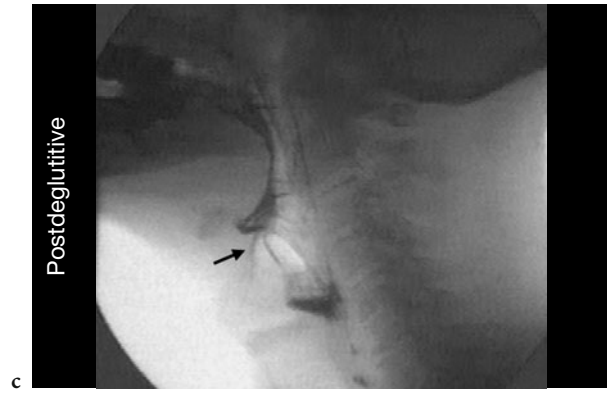


c

Fig. 10.20a–c. a A 65-year-old man after a mild stroke. The opening of the larynx closes too late or in an incomplete manner. In this case contrast medium enters the subepiglottic space (*arrow*). It may penetrate deeper into the supra-glottic space (*arrowhead*). **b** When this relatively mild disorder of closure is present, most of the material that enters is pressed back into the pharynx from the larynx (*arrow*). **c** Postdeglutitivly only the subepiglottic space is marked (*arrow*)

Pre-deglutitive Aspiration (Figs. 10.21, 10.22)

Intra-deglutitive Aspiration. The second important timing of aspiration is intra-deglutitive aspiration, which occurs during the rapid involuntary act of swallowing (Fig. 10.23).

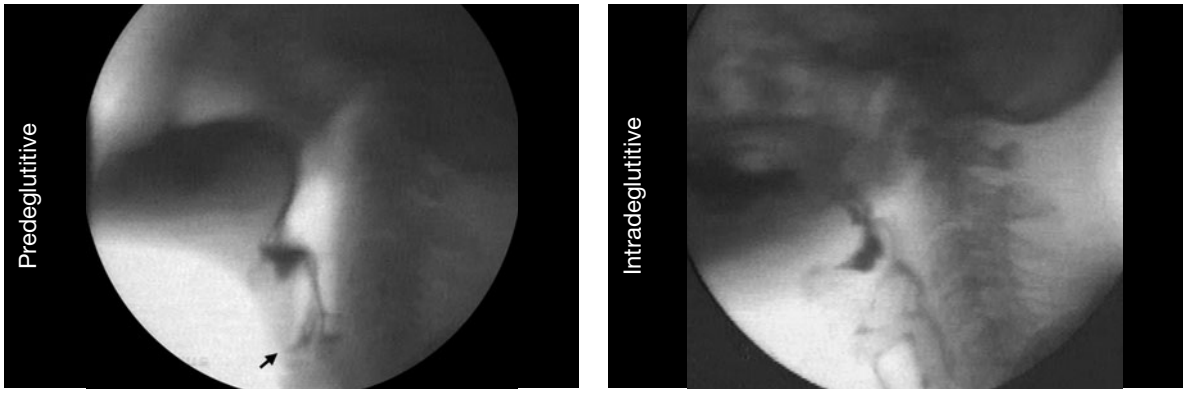
Post-deglutitive Aspiration. The third basic form of aspiration is post-deglutitive aspiration after the involuntary act of swallowing. This form of aspiration occurs during incomplete swallowing, which causes retention in the pharynx (Fig. 10.24). Frequently, it is due to weak pharyngeal muscles.

Severity of Aspiration. An important aspect of the evaluation of aspiration is its severity. Video cinematography can be quite valuable here. However, it should be mentioned that any decision with regard to prognosis and therapy can only be made by taking all clinical data into account.

The simple gradation described in Fig. 10.25 is helpful for reporting the severity of aspiration. Several examples of intra-deglutitive aspiration of various grades of severity are also given in Fig. 10.25.

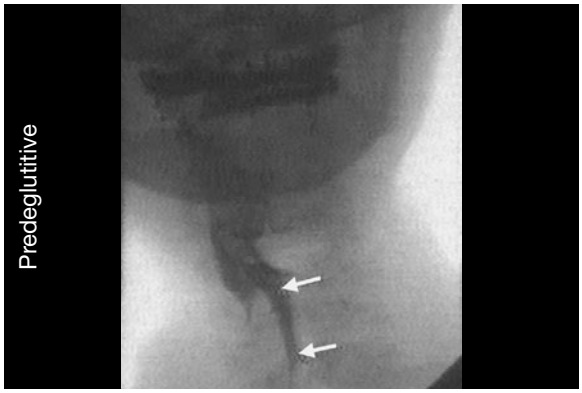
When the coughing attack has subsided or is entirely absent, this always signifies a high grade of aspiration and a great likelihood of broncho-pulmonary complications.

At the end of an examination, the bronchial tree should be documented with chest films or fluoroscopy. This facilitates an estimate of the depth of aspiration and the amount of aspirated material.



a **b**

Fig. 10.21a,b. a This 62-year-old man who experienced a stroke clearly aspirates before the involuntary act of swallowing, as the swallowing act starts too late and contrast medium has entered the larynx long before the initiation of swallowing (*arrow*). **b** Mild aspiration of some drops of water-soluble contrast medium is visible. There is an incomplete epiglottic tilt and retentions in the valleculae



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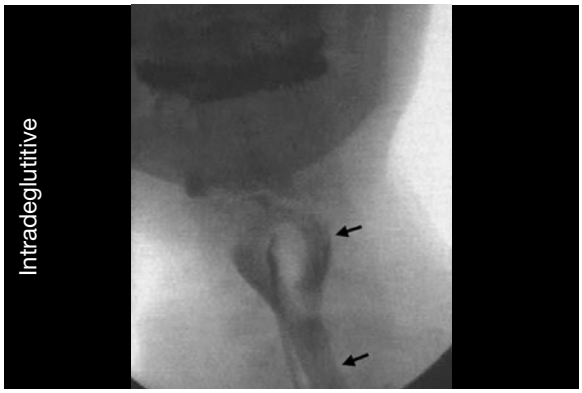
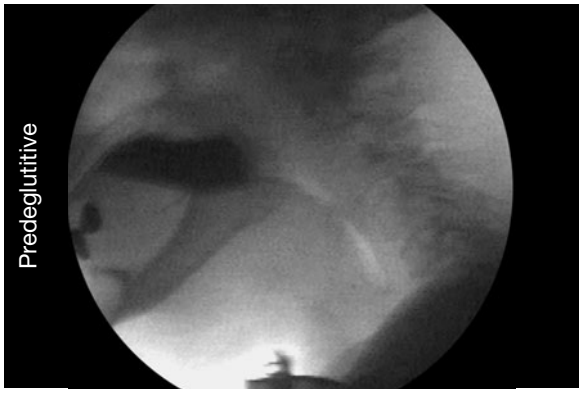


c

Fig. 10.22a–c. a Frontal view of a 64-year-old man with fracture of the skull base and cerebral haemorrhage shows premature leakage and pooling of contrast material in the right piriform sinus (*arrow*). **b** Barium enters the larynx and the trachea before the involuntary swallow (*arrows*). **c** Intradeglutitive pharyngeal propulsion in the frontal projection when the patient tries to clear the retentions with repeated swallows (*arrows*). Less than 10% of the bolus was aspirated before the involuntary swallow



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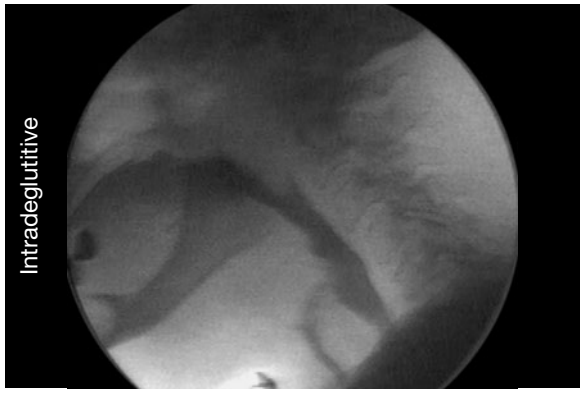


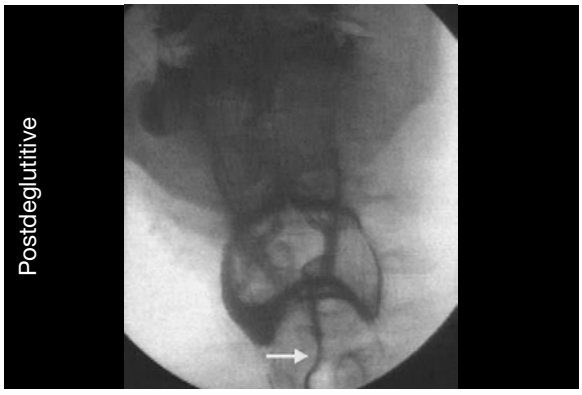
b

c

Fig. 10.23a–c. a This 77-year-old woman after laser resection of a laryngeal carcinoma, presents with a competent seal of the oral cavity dorsally before swallowing. **b** The postoperative defect causes an insufficient closure of the larynx and more than 10% of the swallowed contrast medium is aspirated during the pharyngeal phase. **c** The aspirated contrast medium has reached the bronchial tree. Thus, after the swallow only small amounts are visible in the trachea

a

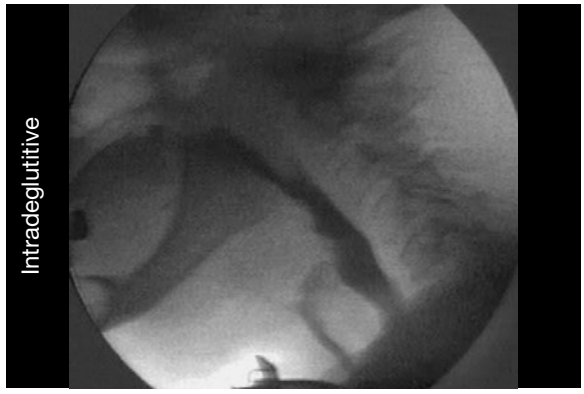
b



c

Fig. 10.24a–c. a A 55-year-old man after a stroke in frontal projection. Pharyngeal retentions in the valleculae and piriform sinuses are present. There is an intense coating of the pharyngeal walls. Aspiration from previous swallows is present. **b** Reduced movements of the lateral pharyngeal walls cannot propel the whole volume of 15 ml through the pharyngeal tube. **c** After swallowing aspiration occurs (*arrow*), caused by retentions in the piriform sinuses. The retentions have overflown the laryngeal entrance, which has opened and moved caudally in the resting phase

a



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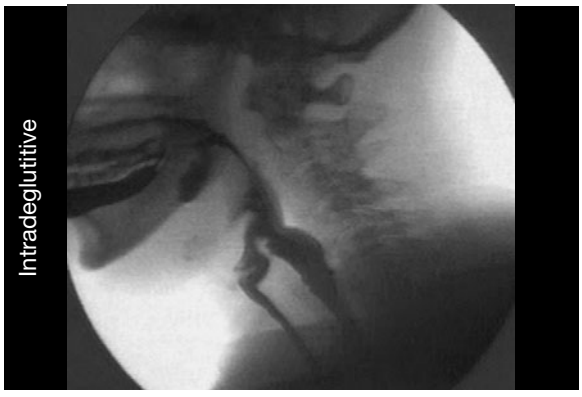


c

Fig. 10.25a–c. a First, a slight grade I aspiration. This means that a few drops of contrast medium are aspirated. **b** Second, a moderate intradeglutitive aspiration, grade II. As far as can be seen, no more than 10 % of the bolus has been aspirated. **c** Third, a massive grade III aspiration with an aspirated volume of far more than 10% of the ingested bolus

10.4.3.5 Pharyngeal Constrictors

Normal Function. The fifth functional unit includes the activity of the pharyngeal constrictors.

The pharyngeal wave of contraction starts at the level of C1 in lateral projection (Fig. 10.26). Here it has advanced up to C2–C3. The rapid upward movements of the hyoid and larynx also take place at the beginning of the involuntary act of swallowing. As the voluntary act smoothly passes into the involuntary one, the physiological beginning may be difficult to determine. In the normal act of swallowing, there should always be a rapid and continuous passage through the pharynx as soon as the angle of the jaw has been passed or the valleculae have been reached (Fig. 10.27).

Delayed Swallowing Reflex. Delayed triggering of the involuntary act of swallowing is an important and common finding (Fig. 10.28). Delay occurs when the contrast medium has reached the level of the valleculae before the involuntary act of swallowing is triggered. Predeglutitive aspiration may be present, when the closure of the laryngeal vestibule is also delayed. Several swallows can show a different length of the delay. Often, the delay is worst in the first swallow and may improve during the examination. An approximated simple measurement can be obtained with the time code of the video recorder or software, by counting the frames from the moment when the contrast material passes the angle of the jaw until involuntary swallow has started. It is clinically useful to count in steps of 0.5 s. Delays from 0.5 s up to 3 s are frequent in neuromuscular disorders. The swallowing reflex is absent if the reflex does not trigger for 30 s.

Symmetric Weakness of the Pharynx. The second important functional disorder of this functional unit, the pharynx, is weakness. It is present when residues of contrast medium remain in the piriform sinuses and the valleculae after swallowing.

The coating of the pharyngeal walls differs with the viscosity of contrast medium. Thus, the high viscosity of barium paste facilitates the detection of pharyngeal weakness (Figs. 10.29, 10.30).

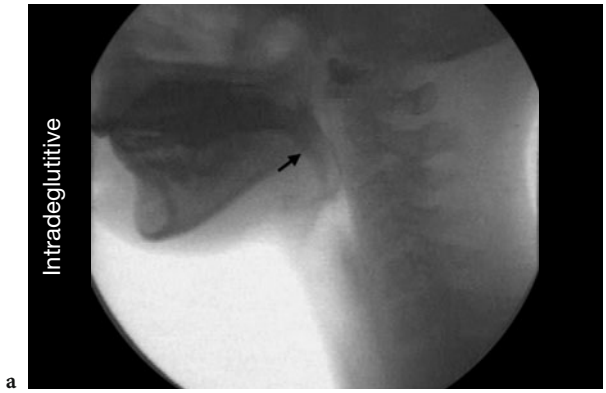
Asymmetric Pharyngeal Weakness. In the case of pharyngeal weakness it is important to distinguish between unilateral or mainly unilateral pharyngeal weakness and a bilateral symmetric pharyngeal weakness, as the therapeutic alternatives differ.

The frontal view is most suitable for radiographic diagnosis of a unilateral pharyngeal weakness, while in oblique views asymmetric retentions can be seen with some experience (Fig. 10.31).

Pharyngocoeles. Pharyngocoeles are usually harmless small out-bulgings of the pharyngeal wall which may become symptomatic when they trap ingested food, which can cause mucosal irritation. Small pharyngocoeles without retentions are common and harmless (Fig. 10.32). In rare cases they can be enormously big (trumpeter).

Space-Occupying Masses. Even large cervical osteophytes can be compensated without severe symptoms. A cerebrovascular accident may lead to a decompensation in such cases. It is often very difficult to decide whether a resection of the osteophytic mass could improve a patient's swallowing function or not (Strasser et al. 2000) (Fig. 10.33).

Space-occupying masses may markedly hinder the pharyngeal passage (Fig. 10.34). Seldomly pharyngeal or laryngeal tumours are detected by videofluoroscopy or spot films, in symptomatic patients, endoscopy is usually performed first.



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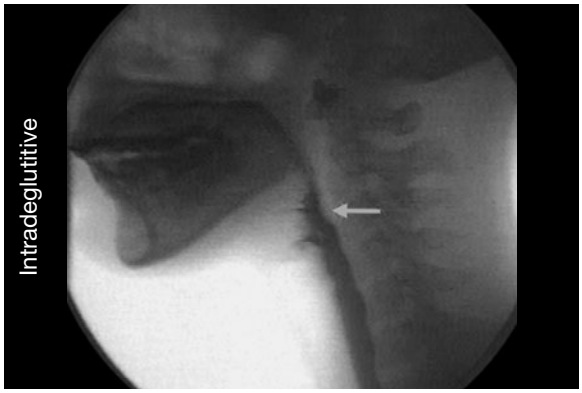

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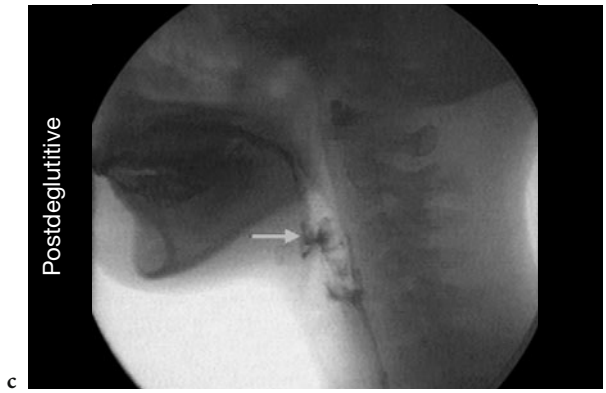


c

Fig. 10.26a–c. a The right time point at which the pharyngeal contraction starts is very important for a physiological swallowing act. If the contrast medium has passed the angle of the jaw or has reached the valleculae, the involuntary act of swallowing should be started (*arrow*). **b** In the lateral view, the onset of pharyngeal wave of contraction can be seen as an indentation (*arrow*), which descends rapidly during the propulsion of the bolus. **c** After the passage, there should only be a coat of contrast medium in the valleculae and the sinus (*arrow*), but no residues with fluid levels

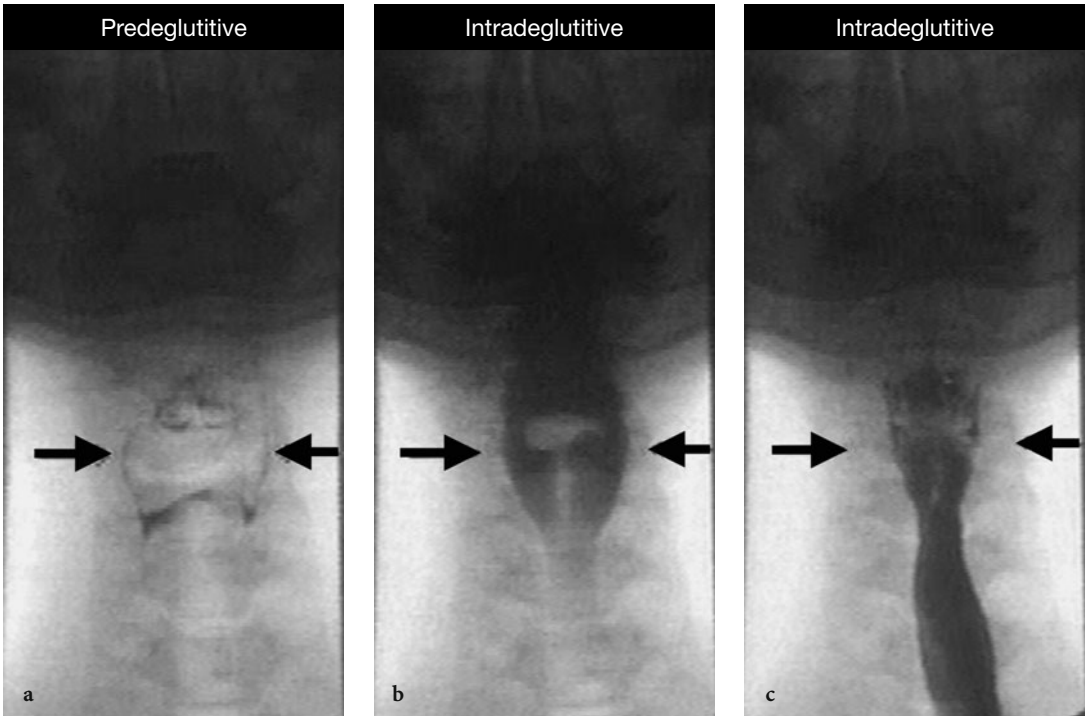
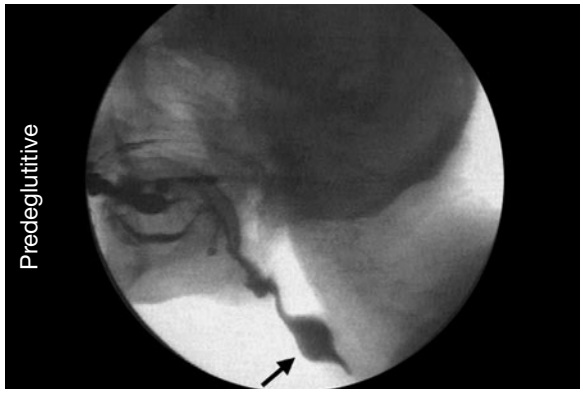


Fig. 10.27a–c. a In the frontal view the pharyngeal walls are delineated lateral convex without retentions in the piriform sinuses (*arrows*). **b** The lateral pharyngeal walls should move rapidly towards medial, with a symmetrical movement (*arrows*). **c** In the late intradeglutitive phase the bolus is squeezed out of the pharynx (*arrows*)



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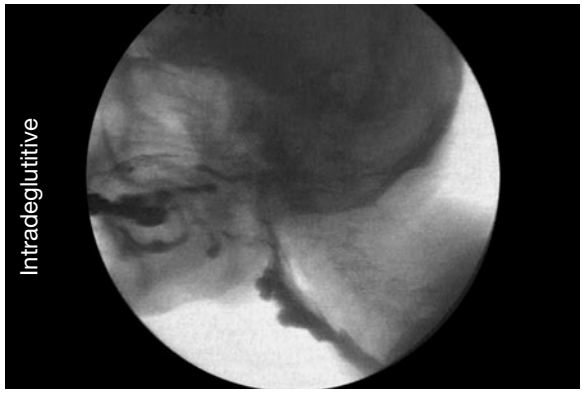

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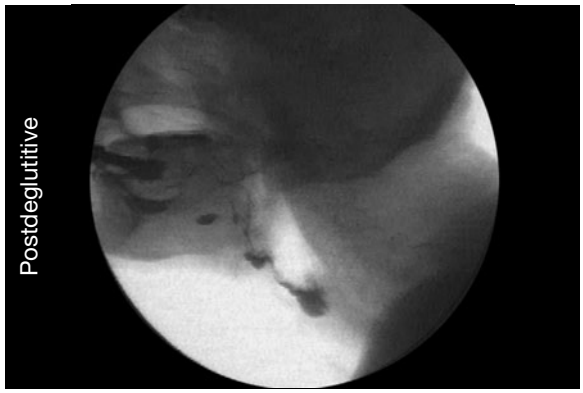
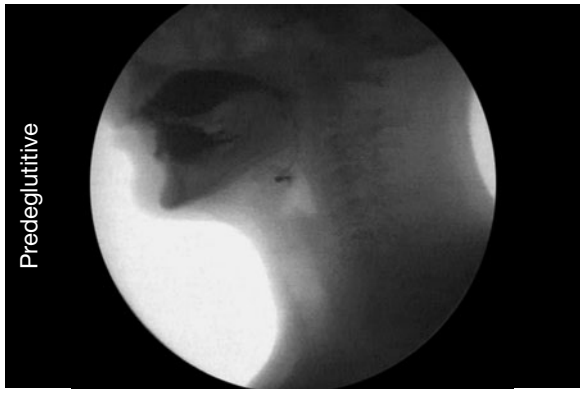


c

Fig. 10.28a–c. a This 86-year-old man after a stroke has markedly delayed triggering. After the contrast medium enters the pharynx, the piriform sinuses (*arrow*) are filled before the involuntary act of swallowing starts. **b** Intradeglutitive phase of the same patient. **c** Retentions in the valleculae and in the hypopharynx after the swallow indicate pharyngeal weakness; no aspiration occurred



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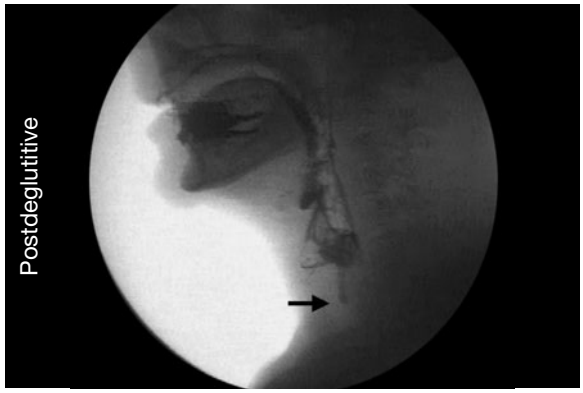


c

Fig. 10.29a–c. a This 66-year-old woman with dermatomyositis has a high-grade pharyngeal weakness. Before the swallow, the bolus is controlled on the back of the tongue. **b** In the late intradeglutitive phase a bolus of 15 ml is separated in two parts (*arrows*). The weak stripping wave is not able to propel the whole bolus into the esophagus, often also combined with a weak tongue movement. Meanwhile, a part of bolus has entered the cervical esophagus. Drops of contrast medium have penetrated into the laryngeal vestibule. **c** Also note the aspiration from retentions after the involuntary swallow – postdeglutitive aspiration (*arrow*)

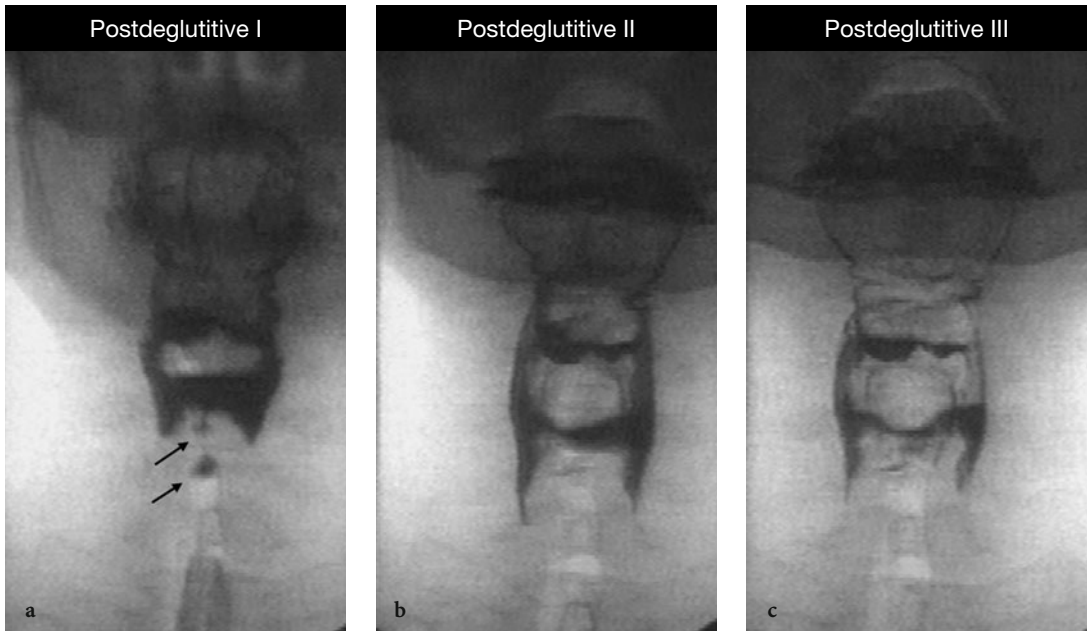


Fig. 10.30a–c. a Same patient as in Fig.10.30 in the frontal projection. Symmetric retentions in the piriform sinuses are present, a fluid level at the laryngeal entrance indicates the potential of postdeglutitive aspiration, contrast material has already entered the larynx (*arrows*). **b** Smaller retentions after a second swallow, which was performed by the patient to clear the hypopharynx from retention. **c** A third swallow succeeded, only slight retention remained, no more material had penetrated into the larynx

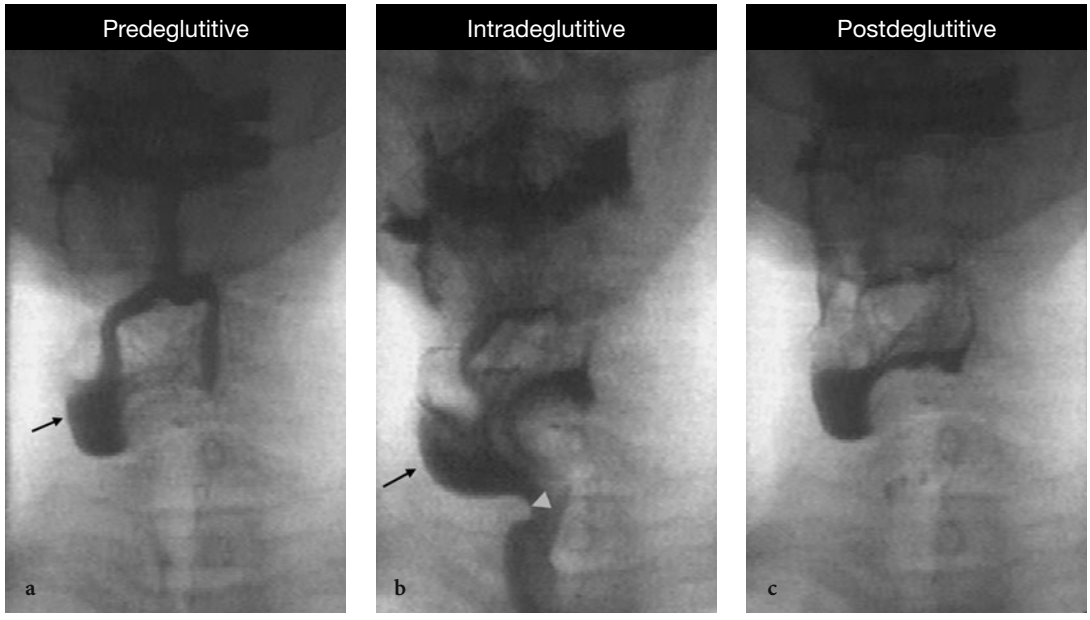


Fig. 10.31a–c. a Ten years before the examination, this 62-year-old woman was operated and radiated on the right side for a floor-of-the-mouth carcinoma. Delayed triggering of the swallowing reflex is present. Contrast material runs over the valleculae into the discrete dilated right piriform sinus (*arrow*). **b** During swallowing the bolus passes over the right side, bulging out (*arrow*) the weak lateral pharyngeal wall. Ipsilateral the pharyngoesophageal sphincter is visible as a lateral cricopharyngeal bar (*arrowhead*). **c** After swallowing one sees the asymmetric or unilateral retention on the right – on the operated side

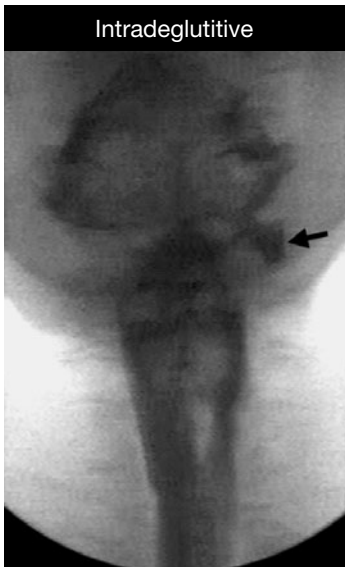
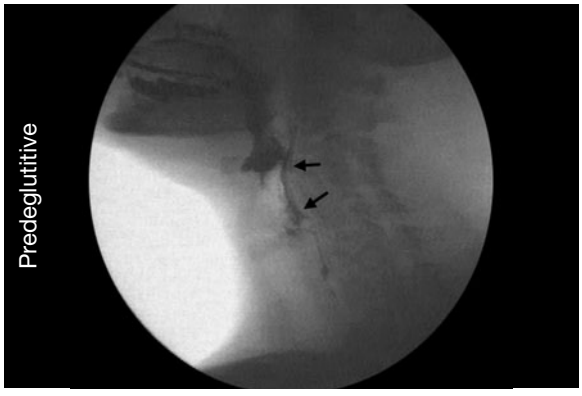


Fig. 10.32. A small pharyngocoele on the left side in an asymptomatic patient. On the left side the pharyngocoele has evaginated (*arrow*)



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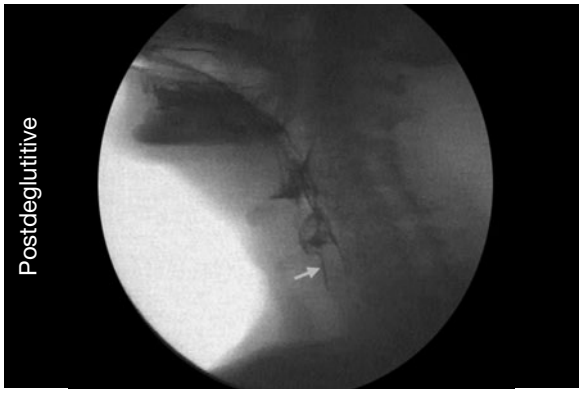


c

Fig. 10.33a–c. a This 63-year-old man has pronounced ventral spondylophytes narrowing the pharynx at the level of the laryngeal entrance (*arrows*). **b** During the swallow the epiglottic tilt is hindered (*arrow*). **c** After swallowing some drops were aspirated from moderate retentions (*arrow*)

a

b

c

Fig. 10.34a–c. a A further example of a pharyngeal space-occupying mass – here we see a recurring tumour of the hypopharynx in a 48-year-old man. The first scene shows broadening of the dorsal pharyngeal soft tissues in a lateral view (*arrows*). **b** In the frontal view one sees the tumour as a structure with irregular margins, in the right portion of the hypopharynx. The passage of the bolus is directed over the left side (*arrows*). **c** Retentions in the pharynx and a moderate postdeglutitive aspiration are present in lateral projection. The epiglottis is thickened (*arrow*) by tumour infiltration. The same appearance of a swollen epiglottis is found in epiglottitis, for example, postradiation in adults or of infectious origin in children

10.4.3.6 Pharyngoesophageal segment

Normal Function. We will now discuss the sixth functional unit of the swallowing act, namely the pharyngoesophageal sphincter or segment (PE segment). It is closed by its resting pressure between swallowing acts. Thus, it can be identified videofluoroscopically between the air column of the pharynx at rest and the air in the cervical esophagus. The PE segment consists of oblique parts of the inferior pharyngeal constrictor muscle, the cricopharyngeal muscle and parts of the cervical esophagus (Fig. 10.35).

Cricopharyngeal Bar. There are conflicting opinions about the percentage of narrowing allowing the diagnosis of a sphincter disorder, but more than 20%–30% seem to be pathological. Three different patients with varying degrees of incomplete opening of the upper esophageal sphincter are shown in Fig. 10.36.

In the case of mild dyskinesia, the dorsal indentation of the barium is discrete. The indentation of a cricopharyngeal bar may occur at various points of time the passage of contrast medium. Furthermore, the indentation may be present for a varying period of time.

Gaping of the PE Segment. In cases of severe neuromuscular disease, the upper esophageal sphincter may reveal a gap, resulting from weakness. The resting pressure is no longer sufficient to close the sphincter (Fig. 10.37).

Web. The next is a morphological finding that is best seen on dynamic recordings. So-called membrane flaps or webs are solitary or multiple thin mucous membranes, most frequently located in the anterior wall of the upper esophageal sphincter. They are usually seen briefly on a few images (Fig. 10.38).

Zenker's Diverticulum. In the region of the pharyngoesophageal junction we find the clinically significant Zenker's diverticulum, which may substantially hinder bolus passage, depending on its size (Fig. 10.39).

Small Zenker's diverticula or pseudodiverticula in combination with a sphincter dyskinesia may also cause complaints (Fig. 10.40).

a

b

Fig. 10.35a,b. a When the contrast bolus reaches the level of the PE segment, the dorsal pharyngeal wall should have smooth margins, because the sphincter relaxes. A slight wave-like margin is normal - it is caused by the intervertebral disks (*arrows*). **b** After the passage of a normal sized bolus no residuals should be left in the pharynx, only coating of the pharyngeal and pharyngoesophageal wall is seen. Radiographically, a posterior indentation at the pharyngoesophageal junction during bolus passage indicates the level of the cricopharyngeal muscle, but may be seen also when the PE segment is closed (*arrow*)

a

b

c

Fig. 10.36a–c. a A 45-year-old woman with globus sensation and mild reflux disease for 3 years. The functional appearance of the sphincter is abnormal. There is a dorsal, rounded impression of the column of contrast medium at the level of the PE segment, representing a moderate cricopharyngeal bar with a narrowing of 30% (*arrow*). **b** A 63-year-old man with dysphagia. Marked dysfunction of the PE segment, a cricopharyngeal bar shows 60% narrowing (*arrow*). **c** Extreme incomplete opening. We find two indentations in terms of a double sphincter, which can be found in various neuromuscular disorders. This 78-year-old woman suffers from a muscular dystrophy

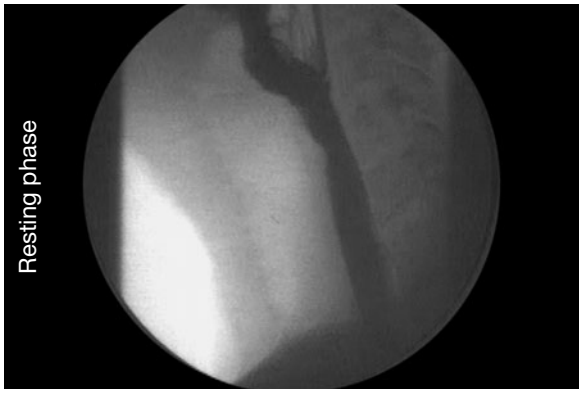
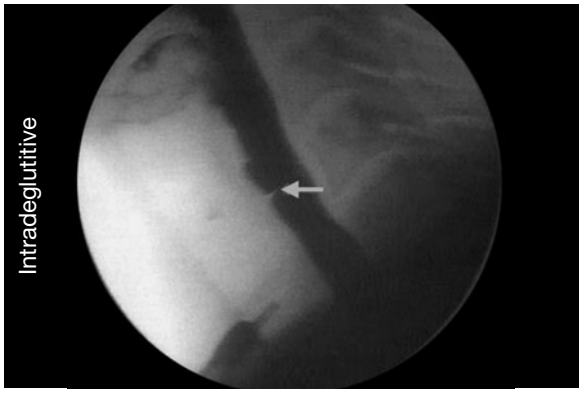


Fig. 10.37. In this 55-year-old woman with mixed connective tissue disease, the PE segment remains open during all the three phases of swallowing. In addition, one always finds a weakness of the pharynx and retentions (*arrows*) in such patients



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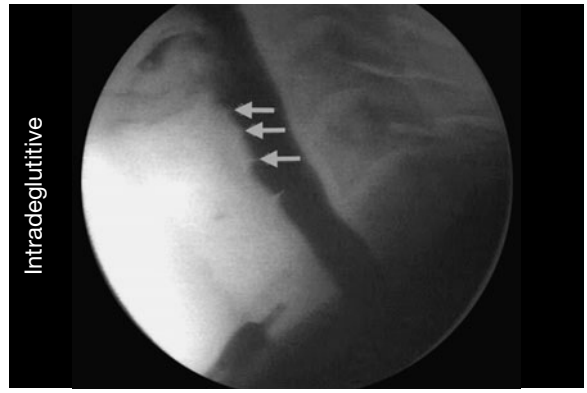

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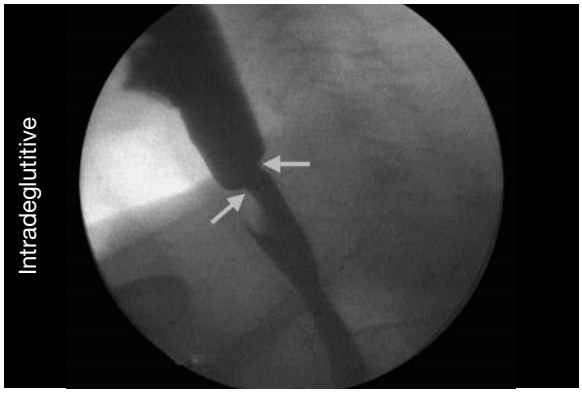

c

Fig. 10.38a–c. a This 38-year-old man suffers from a globus sensation. Small web located ventrally in the lower PE segment (*arrow*). The web is only visible for parts of a second. The small membrane flap will hardly hinder the passage, yet is an indirect indication of other accompanying functional disorders. **b** Typical venous plexus at the postcricoid level. This normal dynamic appearance of an inconstant irregularity must not be misdiagnosed as a web (*arrows*). Webs are always thin and sharply delineated. This differentiates them from the retrocricoid venous plexus, which shows normal, movable, round mucosal folds. **c** This 81-year-old man with dysphagia for solids has a circular web with a marked jet phenomenon. The obstruction by a web may accelerate the flow after the narrowing. This functional finding is known as the "jet phenomenon"

a

b

c

Fig. 10.39a–c. a A 77-year-old woman with high-grade dysphagia for solid and liquid foods. Here we see a constant Zenker's diverticulum, a little more than 2 cm in size. Even without contrast medium it can be detected by a small air bubble in the dorsal soft tissues of the pharynx (*arrow*). **b** Despite its small size of 12 mm, this diverticulum (*arrow*) causes a compression of the gastrointestinal pathway and hinders the bolus passage. **c** After swallowing a constant 12 mm diverticulum is left

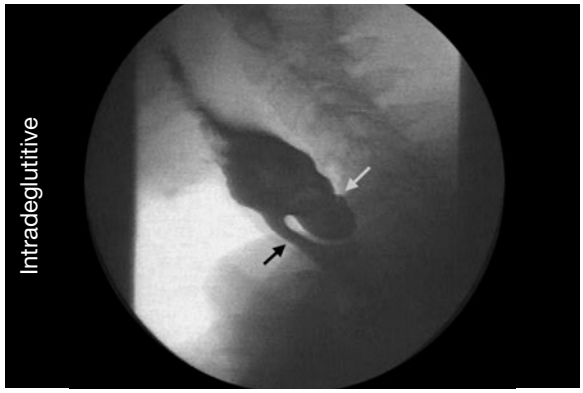

Fig. 10.40. This 73-year-old woman with massive dysphagia has a Zenker's diverticulum approximately 4 cm in size, seen on lateral view. Very large Zenker's diverticula are usually visualised as obstructions of passage even on ordinary static images. On dynamic images, we see here an incorrect esophageal pathway – displayed by the characteristic ridge between the diverticulum and the esophagus. The point of entry into the diverticulum (*white arrow*) is markedly wider than the esophageal pathway (*black arrow*). Besides, the latter is compressed by filling of the diverticulum

10.4.3.7 Esophagus

Normal Function. The esophagus, the seventh functional unit, is very different from the others. The esophageal passage takes about 10 s – about ten times the duration of the pharyngeal passage. The examination technique is also different, as one moves the device along with the slow peristaltic wave of the tubular esophagus from cranial to caudal. The device is kept stationary only at the beginning and the end of the esophageal passage. The analysis is focussed on the dynamic movements of the esophageal walls (Figs. 10.41, 10.42).

Delay of Transport in the Erect Position. One should keep in mind, that pseudoachalasia [tumour stenosis of the esophagogastric junction (EGJ)] cannot be differentiated videofluoroscopically from achalasia with certainty – endoscopy has to be performed (Figs. 10.43, 10.44).

Delay of transport in the Horizontal Position. The most frequent passage disorder is delayed esophageal transport in prone or supine position. In oblique, prone RAO position, a single swallow of 10 ml is partially transported (Figs. 10.45, 10.46).

Non-propulsive Contractions. Non-propulsive or tertiary contractions are more or less strong constrictions of the esophagus that occur in addition to the wave of contraction (Fig. 10.47). They are usually combined with a proximal escape or a support level.

Esophageal Stenoses. Stenoses of the esophagus, which are usually easier to diagnose on dynamic than on static X-ray images, can be tested for their diameters (Fig. 10.48). Placebo tablets of 13–15 mm diameter allow an exact measurement of the esophageal width. Furthermore, the tablet may produce symptoms which reproduce the patient's complaints.

If standard boluses of 10 ml do not obtain a clear delineation of the EGJ, about 150 ml of very thin barium can be applied by a straw – in the horizontal position. The patient may be advised to swallow repeatedly. This causes intradeglutitive inhibition of the peristaltic waves and the esophagus starts to fill up in monocontrast. This can help to diagnose subtle stenoses and rings as an adjunct or alternative to a placebo tablet (Fig. 10.49).

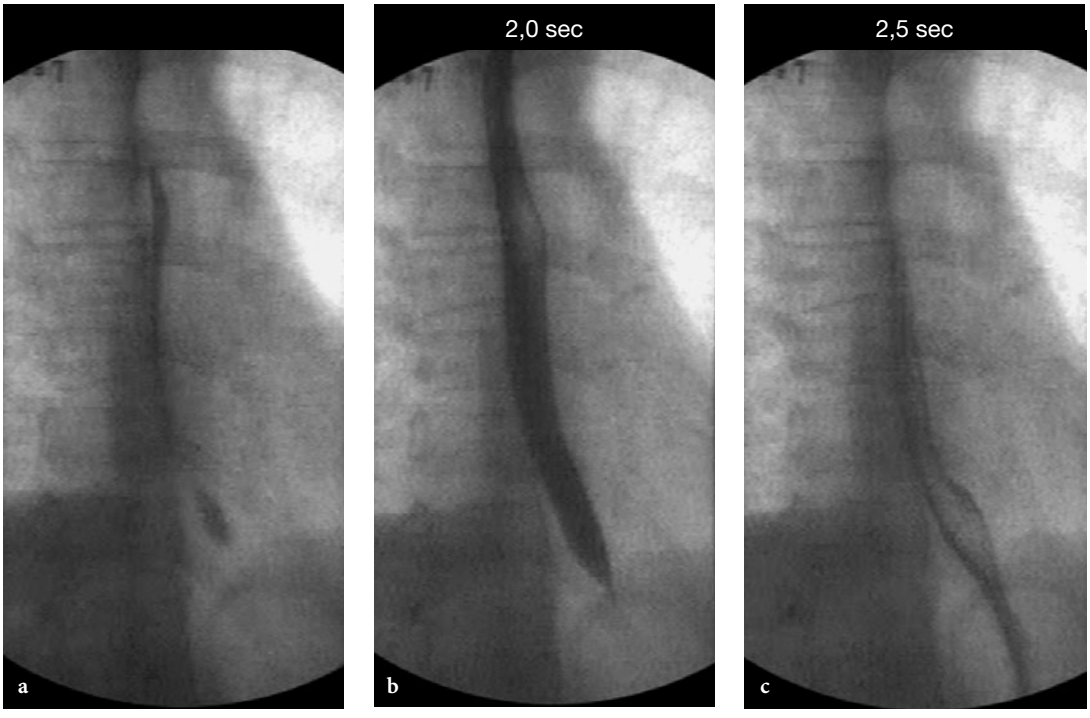

Fig. 10.41a–c. a Left oblique projection of the lower esophagus before the esophageal phase. **b** At 2 s after initiation of the pharyngeal swallow. It is important here to observe the timely, rapid emptying that occurs; 10 ml of barium should be able to pass immediately. The esophageal passage in standing position takes place much faster than that in horizontal position – because of gravity. **c** At 2.5 s later, the bolus has passed. This is normal when the bolus passes slowly through the esophagogastric junction. However, "10 ml barium – 10 seconds" is a basic approximation of normal esophageal transport, in the erect and horizontal position (Schima et al. 1992). If the bolus takes between 5–10 s, a fluid level occurs for this short time span as a normal finding

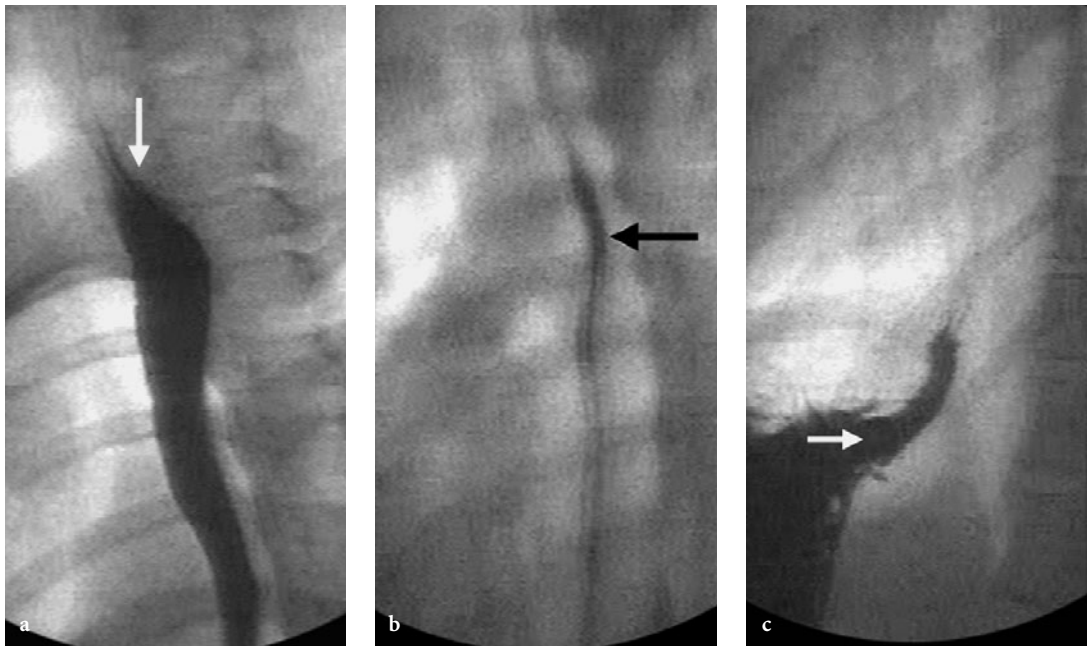

Fig. 10.42a–c. a A normal esophageal passage in right prone anterior oblique position with the left shoulder raised. At the beginning of the esophageal phase a cranially V-shaped upper end of the bolus (*arrow*) defines the peristaltic contraction, which pushes the bolus caudally at a speed of 4 cm/s. Additionally, this view provides excellent delineation of the PE segment during the pharyngeal phase. **b** The bolus is followed through the middle third of the esophageal tube. Small residues of contrast medium that remain at the level of the aortic arch after the passage are normal (*arrow*). Radiologically detectable motility disorders are mainly depicted in terms of delayed passage, abnormal wall movements and structural lesions. Delayed passage is by far the most common radiological functional finding in the upper gastrointestinal tract. **c** 10 ml of barium suspension are transported into the stomach at the end of the esophageal phase

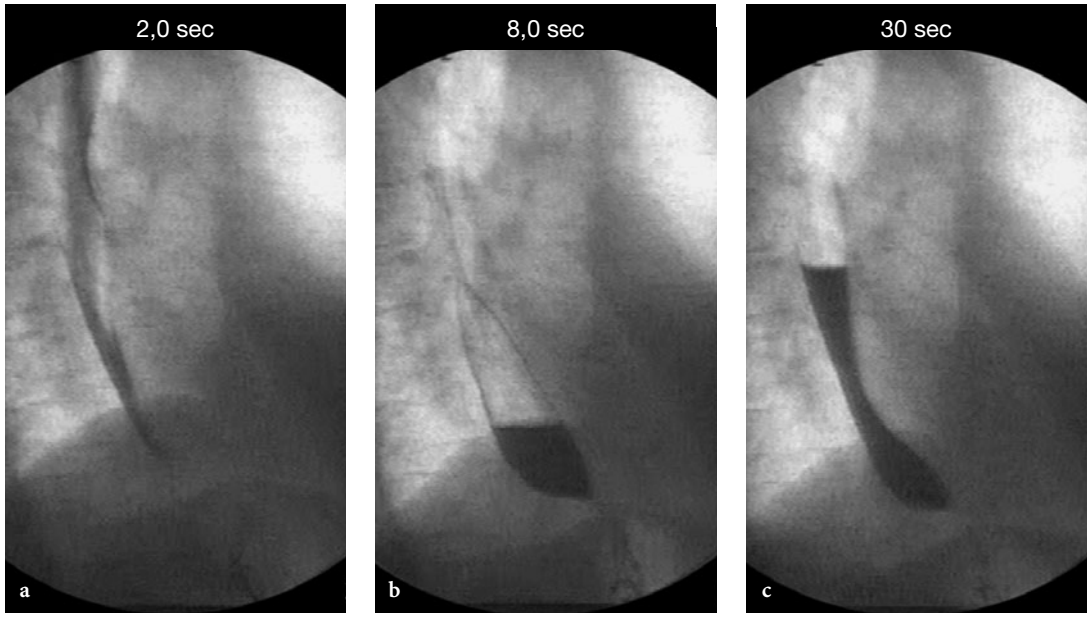

Fig. 10.43a–c. a This 65-year-old woman suffers from achalasia of the lower esophageal sphincter. At 2 s after the pharyngeal phase, parts of the bolus reach the esophagogastric junction. **b** At 8 s after onset of swallowing, a little fluid level remains above the esophagogastric junction (EGJ). **c** The passage of 10 ml of barium takes more than 40 s in this case. The persistent fluid level in the distal esophagus, which is characteristically seen with the patient in standing position, moves up and down without completing the passage. This radiographic sign is also known as a "support level". To establish a delayed esophageal transport in the erect position is a "must" when the esophagus is examined. Many patients with a symptomatic achalasia without a marked dilatation of the tube like in this case, are reported as normal, since this valuable sign is not known by many radiologists. A manometry should be performed in such cases

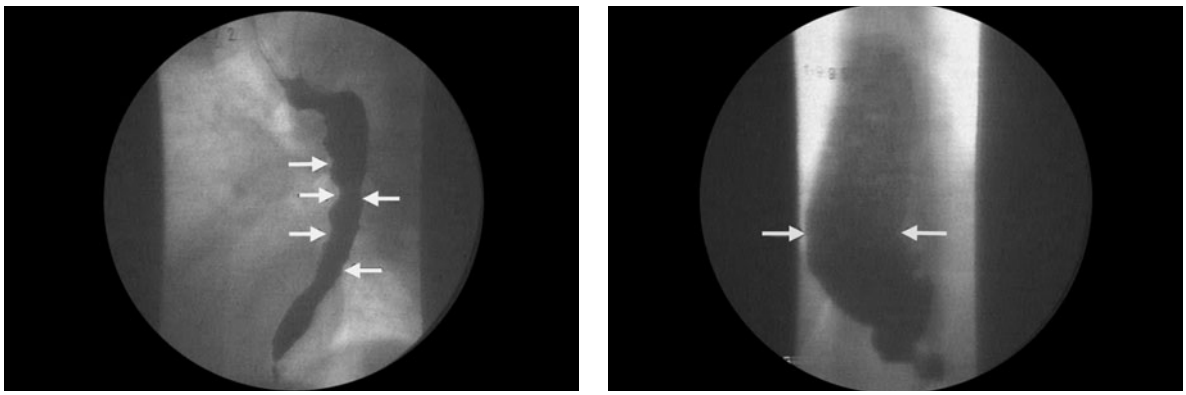

Fig. 10.44a,b. a This 78-year-old man suffers from achalasia. In severe achalasia a support level persists during the entire examination. Only small quantities of contrast medium are able to pass through a beak like esophagogastric junction. In this case we also see strong non-propulsive contractions – a further important functional finding (*arrows*). **b** If the dilatation of the esophagus is severe (*arrows*), the diagnosis of achalasia is almost always reported on simple esophagrams. Note the inhomogeneous appearance of the material within the massively dilated esophagus. This is due to undigested food, which did not pass into the stomach

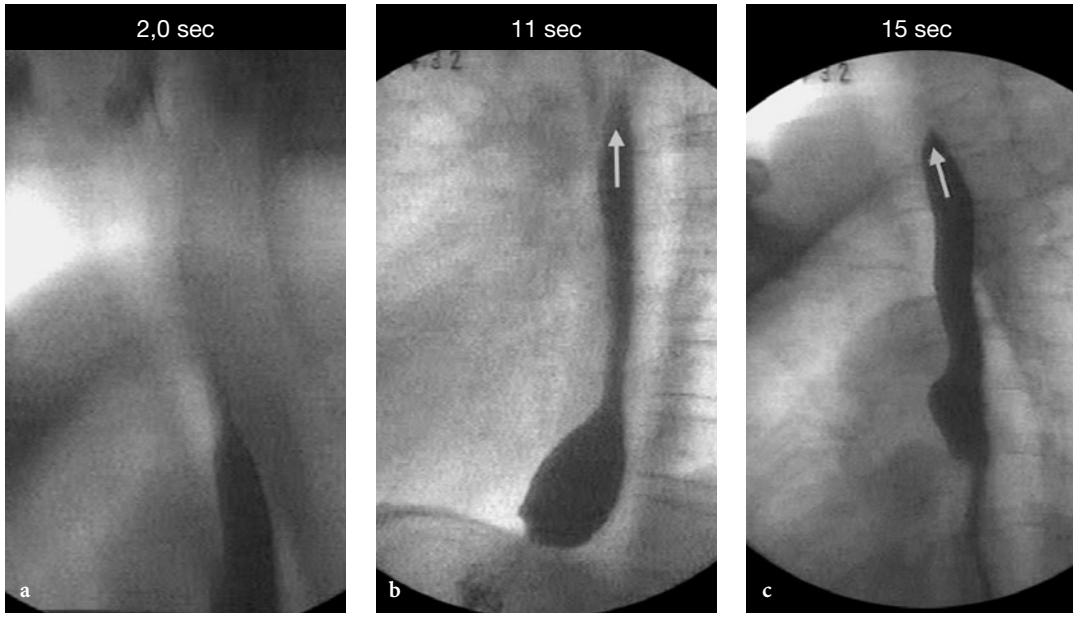

Fig. 10.45a–c. a In this 65-year-old woman with achalasia we have already seen the support level (see Fig. 10.44) while standing. In prone position, the contrast medium is initially transported. **b** Having reached the EGJ the contrast medium now flows back in a proximal direction, because the esophageal contraction is too weak to propagate 10 ml of barium. This indirect sign of reduced contraction strength is known as "proximal escape". **c** The contrast material has reached the upper esophageal sphincter. Contrast medium has flown back to the upper esophageal sphincter. In gastroesophageal reflux or even as a side effect of various medications, one frequently finds a motility disorder of the esophagus

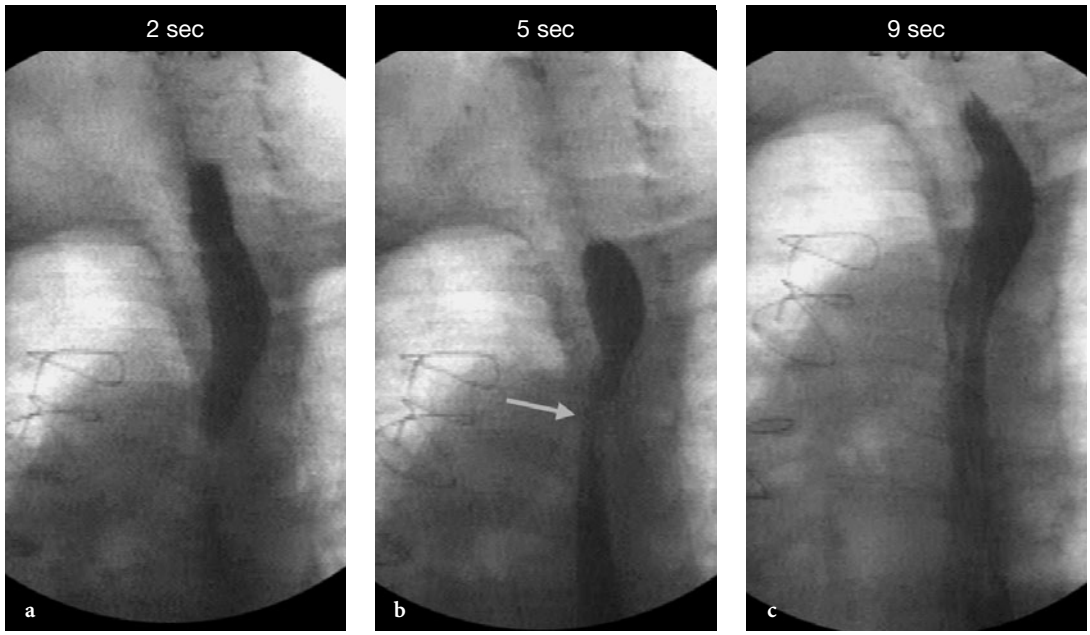

Fig. 10.46a–c. a This 66-year-old patient after a bypass operation takes several different medications for his heart and suffers from reflux with mild dysphagia since a long time. The esophageal passage in prone position has already started. **b** The bolus is segmented (*arrow*), the upper part of the bolus is left by weak esophageal contraction. **c** After the incomplete clearing of the bolus, the esophageal tube is filled with the contrast material, which had escaped proximally to the weak descending wave of primary peristaltic contraction

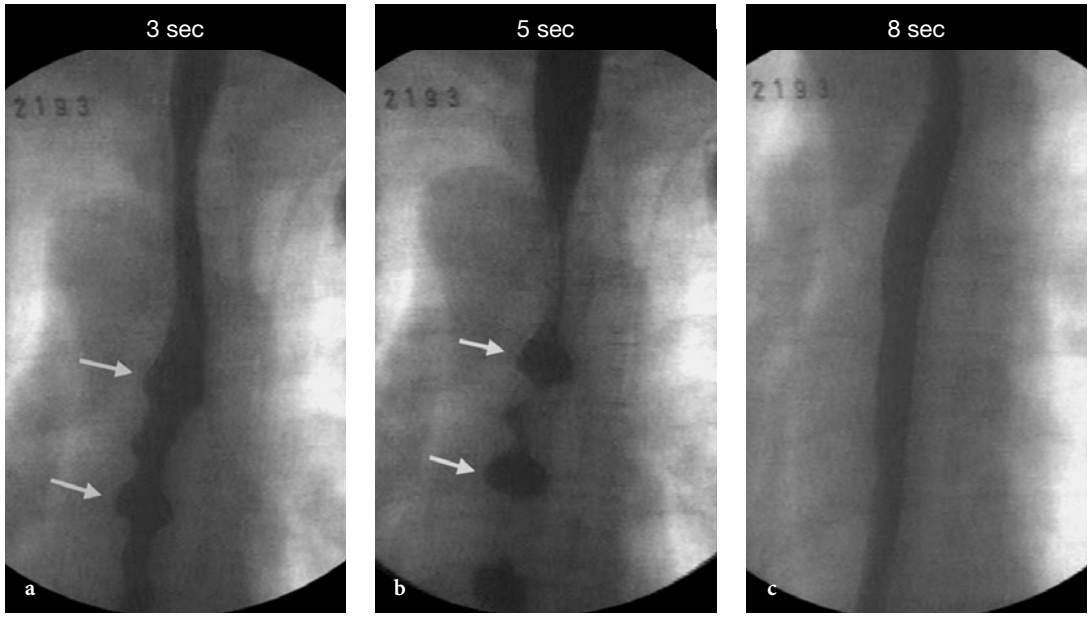

Fig. 10.47a–c. a This 53-year-old man who has suffered from severe dysphagia for solids for several years has entirely constricting, non-propulsive contractions. Three seconds after the onset of the pharyngeal swallow, between the non-propulsive contractions, round segments of the tube do not contract (*arrows*). **b** Two seconds later the non-propulsive contractions have separated the non-contracting segments – "cork screw" or "rosary bead" esophagus are well known, but are non-specific descriptions of this severe kind of esophageal motor disorder. Manometry is the gold standard to classify motor disorders of the esophagus. **c** Three seconds later, the esophagus shows smooth margins again. In patients with long-lasting motor disorders, the non-propulsive contractions may be shown constantly during all phases of swallowing

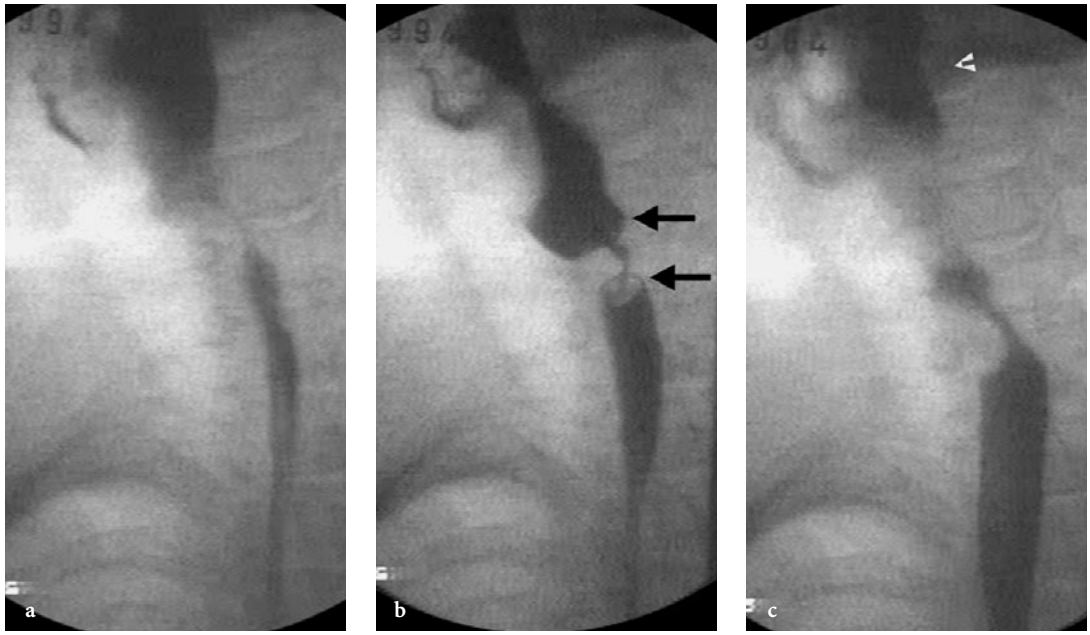

Fig. 10.48a–c. a A 55-year-old woman with dysphagia and a carcinoma of the proximal esophagus. An endoscope could not be advanced, videofluoroscopy was the first diagnostic test. Note that the carcinoma is invisible at the early esophageal phase. **b** For only 0.5 s, the narrow malignant stenosis is depicted clearly (*arrows*). **c** Some frames later, the lesion is obscured. The dynamic investigation is especially advantageous in this setting, the lower spatial resolution of videofluoroscopy is compensated by the extraordinarily useful time resolution, when esophageal stenosis has to be diagnosed. At a frame rate of 3-4 spot films per second, digital recording proves to be an alternative technique for structural lesions, but not for dynamic evaluation of motility

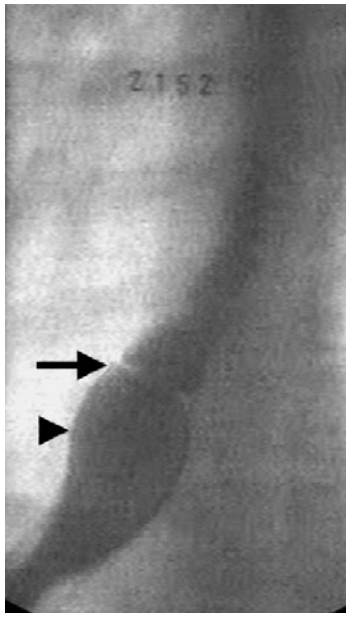

Fig. 10.49. The most common stenosis of the esophagus is the Schatzki ring. A typical Schatzki ring in a 30-year-old man with dysphagia for solids (*arrow*). The ring is located exactly at the gastroesophageal junction and is nearly always combined with a hernia (*arrowhead*). It is crucial to investigate the patient prone and supine, since rings and hernias of the EGJ may be seen in prone or supine position only. The Schatzki ring acts as a diaphragm and can cause impaction of solid food. During bolus passage the ring is only visible for a second or two, depending on the bolus volume

Esophageal Diverticula. Diverticula of the upper digestive tract are an important diagnosis (Fig. 10.50). Whereas the Zenker's diverticulum may be easy to diagnose, the mid-esophageal and epiphrenic diverticula can be obscured by severe non-propulsive contractions. The esophageal tube above and below a mid-esophageal diverticulum shows invariably a delayed esophageal transport and non-propulsive contractions. Incidentally, mid-esophageal diverticula may be found in non-dysphagic elderly people. It is crucial for further treatment, regardless of whether food impaction at the level of the diverticulum is present or not. Furthermore, it is important to note that epiphrenic diverticula are almost always combined with achalasia of the lower esophageal sphincter.

Esophagogastric Junction. In 80% of patients with reflux disease a hiatal hernia is present, but only 50% of patients with hernia suffer from reflux disease. The radiographic diagnosis of a small hiatal hernia is not of specific clinical importance, nevertheless, the radiographic examination of the esophagogastric junction is the method of choice to obtain pertinent topographic information (Figs. 10.51–10.54).

The radiologic contributions to suggest reflux disease by means of functional observations, like reflux or the dynamic appearance of the EGJ are controversial. This chapter does not intend to discuss provocative tests for reflux, like the water-siphon test. Selected observations of authors, who had been interested in the dynamic appearance of the EGJ associated with gastroesophageal reflux will be mentioned briefly. Reporting on the dynamic EGJ can enrich the view of videofluoroscopy and suggest further work up with pH monitoring or endoscopy. Topographic information can also help in planning surgical therapy.

Most patients with heartburn can be managed symptomatically. With persistent, atypical symptoms investigation may be required. The dynamic evaluation can diagnose the presence of a hernia, its topographic relations, abnormal esophageal peristalsis, cricopharyngeal dysfunction and suggestive observations, like the cardia rosette, the angle of His and the width of the EGJ (Figs. 10.55–10.57).

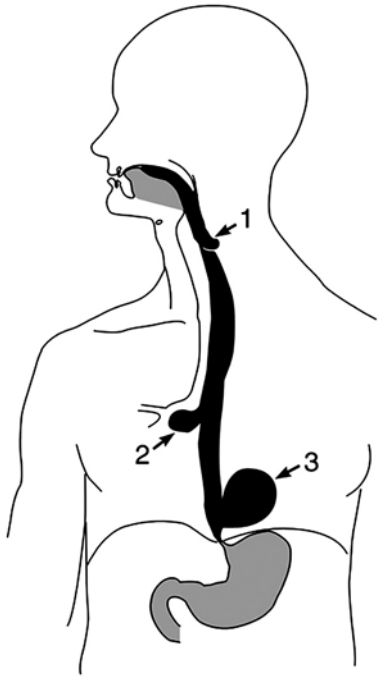

Fig. 10.50. *1*, Zenker's diverticulum; *2*, mid-esophageal diverticulum; *3*, epiphrenic diverticulum

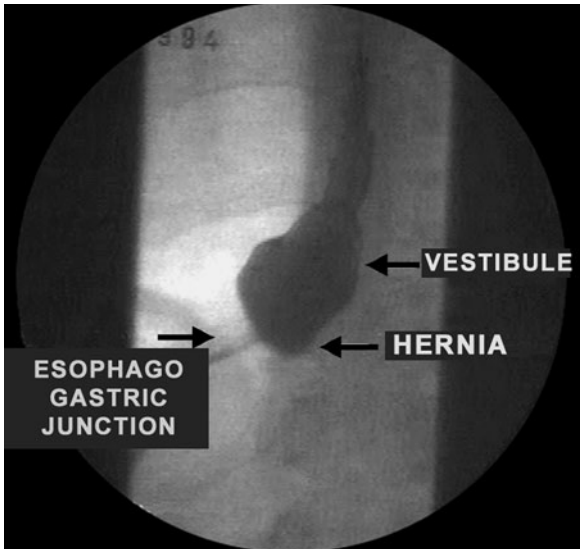

Fig. 10.51. The esophageal vestibule, a slight broadening of the gut distal to the muscular A-ring should end at the level of the diaphragm (see Fig 10.56a). A hiatal hernia can be diagnosed when the vestibule ends at least 2 cm above the EGJ. The recess between the vestibule and the EGJ may be diagnosed as a part of the stomach, protruding upward into the thorax – a hiatal hernia

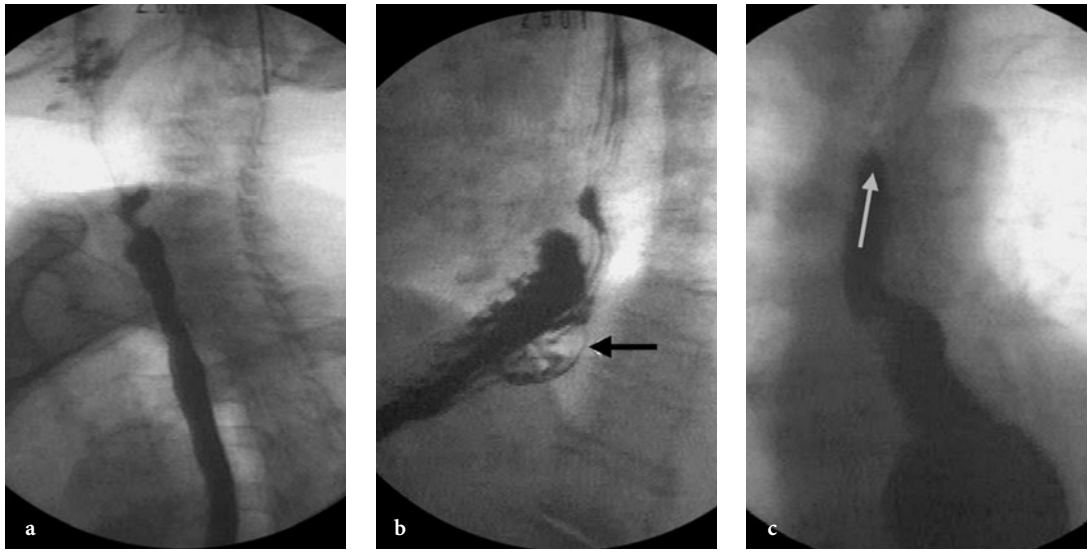

Fig. 10.52a–c. a A 73-year-old woman with heartburn and mild dysphagia. In the prone right anterior oblique position a cricopharyngeal bar is visible. **b** The end of the esophageal phase reveals an about 7-cm large hiatal hernia. **c** Turning the patient supine gastroesophageal reflux to the upper third of the esophagus was present. pH monitoring is the gold standard for directly measuring reflux. However, if reflux to the upper esophagus can be demonstrated, a further clinical work up is recommended

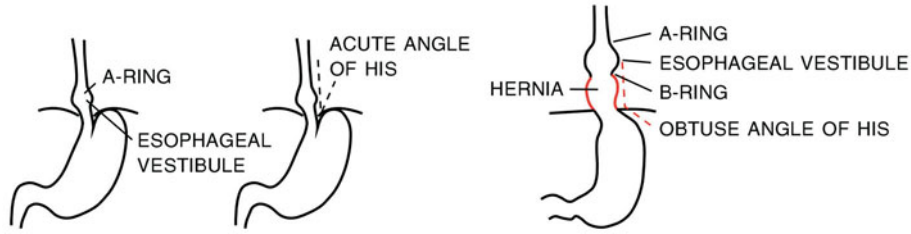

Fig. 10.53. The two graphs on the left represent the normal appearance of the esophagogastric junction, the graph on the right demonstrates an axial hernia and an obtuse angle of His (Fujiwara et al. 1998; Munzer 1997)

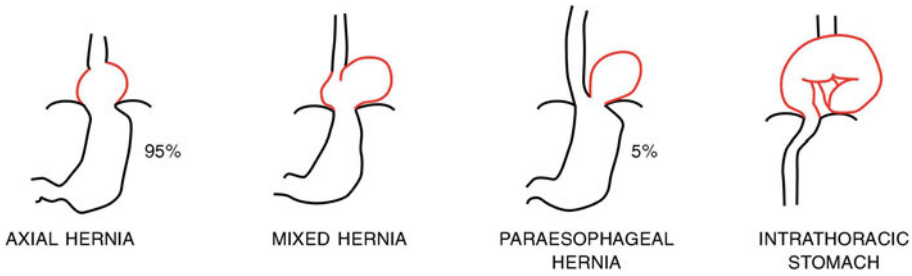

Fig. 10.54. Axial hiatal hernia is common. When a part of the hernia reaches more cranially than the EGJ, it is called a "mixed hernia". Paraesophageal hernia is characterized by an EGJ at the level of the hiatus and a hernia, which enters the thorax separately left and ventrally to the EGJ. An intrathoracic or upside-down stomach means a complete displacement of the stomach into the mediastinum

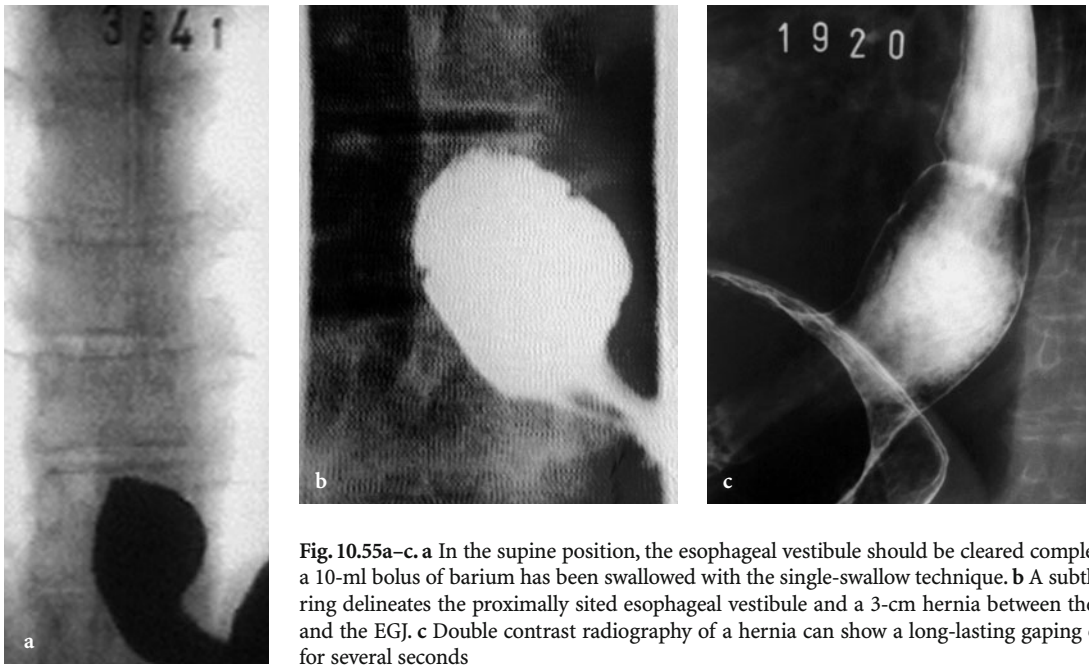

Fig. 10.55a–c. a In the supine position, the esophageal vestibule should be cleared completely when a 10-ml bolus of barium has been swallowed with the single-swallow technique. **b** A subtle mucosal ring delineates the proximally sited esophageal vestibule and a 3-cm hernia between the vestibule and the EGJ. **c** Double contrast radiography of a hernia can show a long-lasting gaping of the EGJ for several seconds

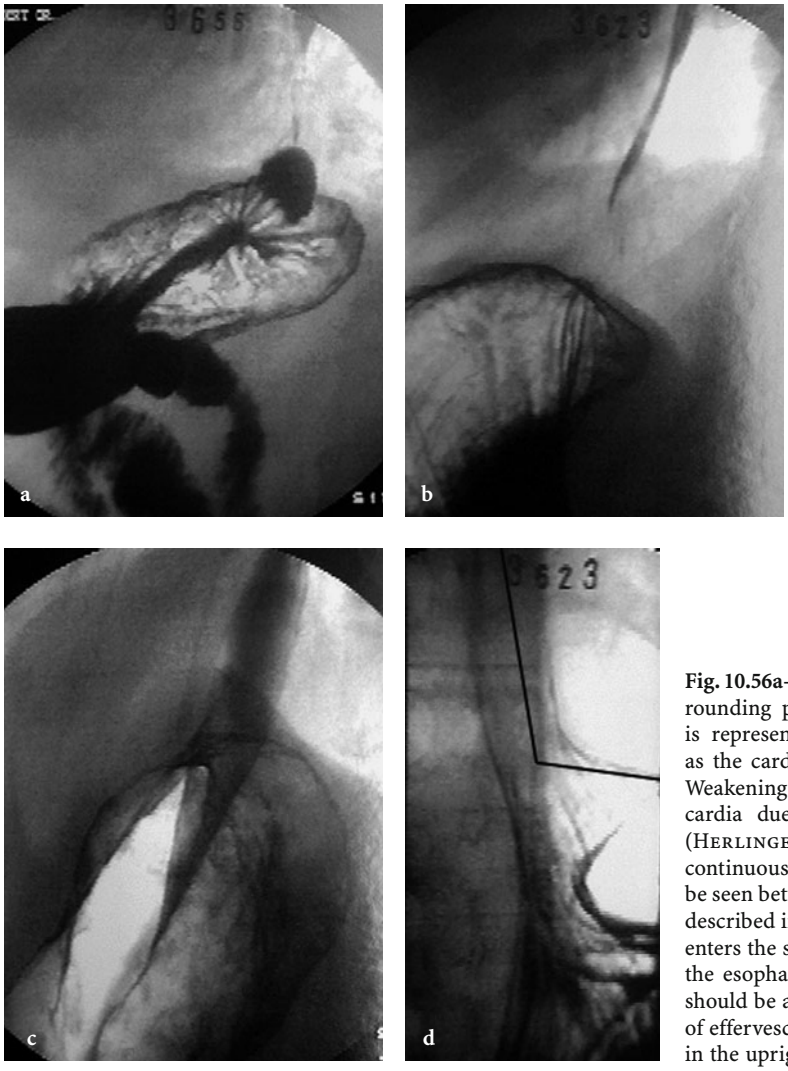

Fig. 10.56a–d. a The cardia is anchored by surrounding phrenoesophageal membrane. The cardia is represented by stellate folds, radiating centrally as the cardiac "rosette" – the normal appearance. **b** Weakening of the ligaments can cause a funnel shaped cardia due to laxity of ligamentous attachments (Herlinger 1980). **c** In severe ligamentous laxity a continuous gaping of the EGJ for some seconds can be seen between the swallows. **d** The angle of His, first described in 1903 is the angle at which the esophagus enters the stomach. The angle is determined between the esophagus and the top of the fornix. The angle should be acute and not obtuse. After the application of effervescent powder, the His angle can be observed in the upright position

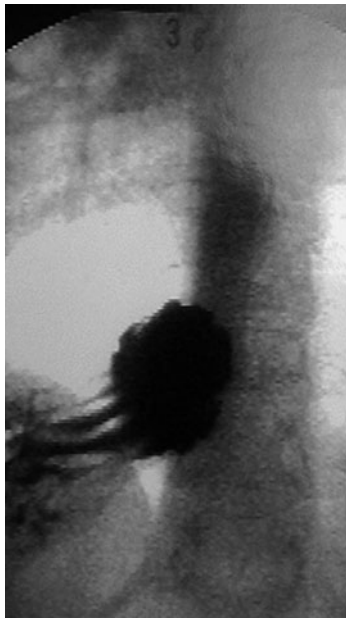

Fig. 10.57. A wide internal diameter of the cardiac esophagus of over 2.5 cm might be abnormal (Graziani et al. 1983)

References

Buchholz DW (1987) Neurologic evaluation of dysphagia. Dysphagia 1:187

Buchholz DW (1996) What is dysphagia? Editorial. Dysphagia 11:23–24

Buchholz DW, Bosma JF, Donner MW (1985) Adaptation, compensation, and decompensation of the pharyngeal swallow. Gastrointest Radiol 10:235–239

Edwards DAW (1974) History and symptoms of esophageal disease of the esophagus. In: Vantrappen G, Hellemans J (eds) Diseases of the esophagus. Springer-Verlag, New York

Ekberg O, Pokieser P (1997) Radiologic evaluation of the dysphagic patient. Eur Radiol 7:1285–1295

Fujiwara Y, Nakagawa K, Kusunoki M., Tanaka T, Yamamura T, Utsunomia J Gastroesophageal reflux after gastrectomy (1998) Possible significance of the angle of His. Am J Gastroenterol 93:11–15

Graziani L, De Nigris E, Pesaresi A, Baldellli S, Dini L, Montesi A (1983) Reflux esophagitis: radiographic-endoscopic correlation in 39 symptomatic cases. Gastrointest Radiol 8:1–6

Hendrix TR (1993) Art and science of history taking in the patient with difficulty swallowing. Dysphagia 8:69–73

Herlinger H, Grossmann R, Laufer I (1980) The gastric cardia in double contrast study: Its dynamic image. AJR 135:21

Jones B, Donner M (1988) Examination of the patient with dysphagia. Radiology 167:319–326

Logemann JA (1995) Dysphagia: evaluation and treatment. Folia Phoniatr Logop 47:140–164

Moser G, Vacariu-Granser GV, Scneider C, Abatzi TA, Pokieser P, Stacher-Janotta G, Gaupmann G, Weber U, Wenzel T, Roden M, Stacher G (1991) High incidence of esophageal motor disorders in consecutive patients with globus sensation. Gastroenterology 101:1512–1521

Munzer D (1997) Angle of His in the cardioesophageal junction: is it a primordial factor in reflux esophagitis? Scand J Gastroenterol 32:847

Palmer JB, Drennan JC, Baba M, (2000) Evaluation and treatment of swallowing impairments. Am Fam Physician 61:2453–2462

Pokieser P, Schober E, Schima W (1995) Videocinematographie des Schluckaktes: Indikation, Methodik und Befundung. Radiologe 35:703–711

Scharitzer M, Pokieser P, Schober E, Schima W, Eisenhuber E, Stadler A, Memarsadeghi M, Partik B, Lechner G, Ekberg E (2002) Morphological findings in dynamic swallowing studies of symptomatic patients. Eur Radiol 12:1139–1144

Schima W, Stacher G, Pokieser P, Uranitsch K, Nekham D, Schober E, Moser G, Tscholakoff D (1992) Videofluoroscopic and manometric evaluation of esophageal motor disorders: Prospective study in 88 symptomatic patients. Radiology 185:487–491

Schober E, Schima W, Pokieser P (1995). Die radiologische Abklärung des Globus pharyngis. Radiologe 35: 724–732

Sonies BC, et al. (1987) Clinical examination of motor and sensory function of the adult oral cavity. Dysphagia 1: 178

Strasser G, Schima W, Schober E, Pokieser P, Kaider A, Denk DM, (2000) Cervical osteophytis impinging on the pharynx: importance of size on current disorders for the development of inspiration. AJR 174(2):449–453

Woo P, Noordzij P, Ross JA (1996) Association of esophageal reflux and globus symptom: comparison of laryngoscopy and 24-h pH monitoring. Otolaryngol Head Neck Surg. 115:502–507

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List of Contributors

Karin Aksglæde, MD
Motility Laboratory
Department of Radiology R
Aarhus University Hospital
Nørrebrogade 44
8000 Aarhus C
Denmark

A. Ba-Ssalamah, MD
Department of Radiology
University of Vienna
Währinger Gürtel 18-20
1090 Vienna
Austria

Jane E. Benson, MD
Assistent Professor
Russell H. Morgan Department of Radiology
and Radiological Science
The Johns Hopkins University School of Medicine
600 North Wolfe Street
Baltimore, MD 21287
USA

Margareta Bülow, SLP, MSc
Departments Neurology and Radiology
Malmö University Hospital
20502 Malmö
Sweden

Michael Y. Chen, MD
Department of Radiology
Wake Forest University School of Medicine
Medical Center Boulevard
Winston-Salem, NC 27157
USA

Christian Czerny, MD
Professor, Department of Radiology
University of Vienna
Allgemeines Krankenhaus
Währinger Gürtel 18-20
1090 Vienna
Austria

Doris-Maria Denk, MD
Professor, Department of Otorhinolaryngology
Section of Phoniatrics
Vienna Medical School
University of Vienna
Währinger Gürtel 18-20
1090 Vienna
Austria

Edith Eisenhuber, MD
Department of Radiology
University of Vienna
Währinger Gürtel 18-20
1090 Vienna
Austria

Olle Ekberg, MD, PhD
Professor, Department of Diagnostic Radiology
Malmö University Hospital
20502 Malmö
Sweden

Peter Funch-Jensen, MD
Department of Radiology R
Aarhus University Hospital
8000 Aarhus C
Denmark

Christian Hannig, MD
Institut für Röntgendiagnostik
Klinikum rechts der Isar
Technische Universität München
Ismaningerstrasse 22
82756 Munich
Germany

Marc S. Levine, MD
Professor, Department of Radiology
Hospital of the University of Pennsylvania
3400 Spruce Street
Philadelphia, PA 19104
USA

Bonnie Martin-Harris, PhD, CCC-SLP
Director, MUSC Evelyn Trammell Institute
for Voice and Swallowing Disorders
Otolaryngology Head and Neck Surgery
Medical University of South Carolina
P.O. Box 250582
150 Ashley Avenue
Charleston, SC 29425
USA

Göran Nylander, MD
Associate Professor
Department of Diagnostic Radiology
Malmö University Hospital
205 02 Malmö
Sweden

Rolf Olsson, MD, PhD
Department of Diagnostic Radiology
Malmö University Hospital
20502 Malmö
Sweden

David J. Ott, MD, FACR, FACG
Department of Radiology
Wake Forest University School of Medicine
Medical Center Boulevard
Winston-Salem, NC 27157
USA

Benoit C. Pineau, MD, MSc. (Epid.), FRCPC
Department of Medicine
Wake Forest University School of Medicine
Medical Center Boulevard
Winston-Salem, NC 27157
USA

Peter Pokieser, MD
Professor, Department of Radiology
Allgemeines Krankenhaus
Währinger Gürtel 18-20
1090 Vienna
Austria

Stephen E. Rubesin, MD
Professor, Department of Radiology
MRI Building 1-08
University of Pennsylvania Medical Center
3400 Spruce St.
Philadelphia, PA 19104-4283
USA

Martina Scharitzer, MD
Department of Radiology
University of Vienna
Währinger Gürtel 18-22
1090 Vienna
Austria

Wolfgang Schima, MD
Professor, Department of Radiology
University of Vienna
Währinger Gürtel 18-20
1090 Vienna
Austria

Rainer Schöfl, MD
4th Department of Internal Medicine
Hospital of the Elisabethinen, Linz and
Department of Internal Medicine IV
Section of Gastroenterolgy and Hepatology,
University of Vienna
1090 Vienna
Austria

Per Thommesen, MD, DMSc
Department of Radiology R
Aarhus University Hospital
8000 Aarhus C
Denmark

Arastoo Vossough, MD
Department of Radiology
Hospital of the University of Pennsylvania
3400 Spruce Street
Philadelphia, PA 19104
USA

Anita Wuttge-Hannig, MD
Gemeinschaftspraxis für Radiologie, Strahlentherapie
und Nuklearmedizin
Dres. Wuttge – Hannig – Rosskopf – Schepp – Sindelar
Karlsplatz 3-5
80335 Munich
Germany

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